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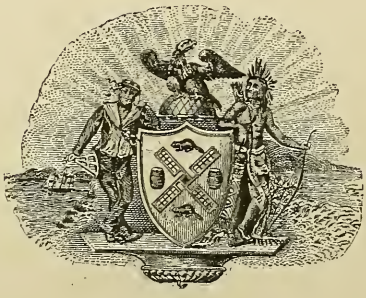

CITY HOSPITAL

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Honorable Robert W. Hebberd,
Commissioner of Public Charities,
New York City:

Dear Sir—The undersigned Editorial Committee, appointed by the Medical Board of the New York City Hospital, beg leave to submit the following Medical and Surgical Report of the Hospital.

In doing so we wish to express our appreciation of the practical support which you have uniformly given the Committee in the work of issuing this, the first Clinical Report to appear from this Institution.

In consulting the records for material for this report the writers have experienced much difficulty owing to the present state of our Index and Filing System. Many important records were imperfect or untraceable. Statistics and conclusions were thus rendered incomplete or unreliable and in consequence had to be omitted. It is the hope of the Committee that this report, issued in spite of these difficulties, will suggest to you what could be done were an adequate filing system instituted. Such a system would enable the patients, the hospital staff and Medicine in general to benefit by the wealth of opportunity to observe the phenomena of diseases and their treatment which this hospital affords. The Committee are of the opinion that an adequate history and filing system properly maintained is essential to good ward work, and they therefore earnestly recommend it to your consideration.

Very respectfully,

C. N. B. Camac, M.D.,
J. A. Fordyce, M.D.,
E. M. Foote, M.D.,
Editorial Committee.

New York City, January, 1909.

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I.—Medical Division

ARTERIO-SCLEROSIS OF THE PANCREAS.

HARLOW BROOKS, M. D.,

Attending Physician.

The following study is based on 75 cases of marked arterio-sclerosis of the pancreatic arteries. These cases were selected as showing a relatively high degree of arterio-sclerosis of the pancreatic vessels as compared with that present in the general or other visceral trunks.

Observation and study of the history of cases which show this condition has convinced me that local arterio-sclerosis is sufficient in many instances to appreciably effect the functions of the pancreas. In a considerable number this change, together with other lesions believed to be secondary, has been sufficiently pronounced to satisfactorily account for some of the more important or striking manifestations shown.

Arterio-sclerosis of the pancreatic vessels is found in a marked degree in the greater proportion of cases which show general arterial disease, and, according to my statistics previously published (American Journal of the Medical Sciences, May, 1906), in point of special involvement of the visceral arteries the pancreatic vessels stand fourth.

Pancreatic disease is now beginning to attract so much attention from clinicians it seems necessary that anatomical studies of so frequent a form of pancreatic disorder should be made, not so much on account of the primary lesion itself, which can be successfully treated in but a small number of cases, but mostly because of the fact that some of the clinically recognizable lesions of the pancreas appear to be primarily dependent on arterial change. This, though suggested by several writers, seems to have been first fully appreciated by Dieckhoff (Beitrage zur Pathologischen Anatomie des Pancreas. Leipzig. 1896).

The arterial supply of the pancreas is derived from several important trunks which give off pancreatic branches a very short distance after their primary origin from the aorta. The body and tail of the organ are chiefly supplied by vessels which arise from the splenic artery. Several descending branches originate directly from the splenic and in addition this trunk commonly gives off a long branch, usually of large size, which runs from left to right in the direction of the pancreatic duct. When this vessel, the arteria pancreatic magna is present, the body and tail of the pancreas are chiefly nourished by it. This trunk I have found to be most commonly and most severely diseased in pancreatic arterio-sclerosis, a fact previously noted by Hoppe Seyler. The gastro-duodenal artery also gives off a branch, the

arteria pancreatis duodenalis superior, supplied to the head of the organ which, in addition, receives the arteria pancreatico duodenalis inferior from the superior messenteric. The head and proximal portion of the body of the pancreas are, however, mostly nourished by a direct branch from the hepatic artery. This vessel stands second to the splenic in its importance and also in relative rate of involvement. These last named vessels, in fact, all those supplied to the head of the pancreas, are less frequently and less extensively diseased in arterio-sclerosis, probably due to their size, abundance and consequent lesser individual importance and also to their anatomical arrangement, permitting abundant anastomosis.

Etiology—The statistics of my cases show that in 19 of the 75, excessive use of alcohol appears to be the most probable etiological factor. Syphilis stands next as the dominant agent in 10 instances and senility in 9. Nephritis of an active character was present in 6 cases, tuberculosis in 4 and acromegalia in 4, 3 of which were complicated by the symptom of glycosuria. In regard to acromegalia I have previously pointed out that arterio-sclerosis and universal fibrosis are essential and early changes in this disease in which they probably appear as a regular reaction to hyper pituituary secretion (Acromegolia, State Hospitals Press, Utica, N. Y., 1899). Five marked examples occurred in conjunction with diabetes mellitus. In the remaining 18 cases the probable etiology varied so as to make classification difficult and in some no satisfactory factors could be determined.

Arterial Lesions—It may seem unnecessary to say at this time, when so much recent work has been reported on arterial disease that no characteristic types of arterial lesions could be associated at all constantly with any specific etiological factor. In one case, which I particularly recall, since I have long used the specimens for demonstration before my class, a marked, almost obliterative endarteritis was present in one segment of the artery and at a distance of but 2 cm. a beautiful example of dilated and atrophied wall with most pronounced changes in the media was shown. Except when the arterial walls are immediately the seat of some such process as gumma formation, the anatomical changes are in no way etiologically characteristic. In this respect the disease of the pancreatic vessels is exactly like the process in general or other visceral vessels. No classification as to etiology on anatomical grounds is yet possible in arterio-sclerosis.

One anatomical condition is, however, quite constant in pancreatic arterio-sclerosis. In nearly every marked instance the pancreatica magna is very much contorted and in some cases lies in many compactly convoluted folds. This condition is also occasionally observed in the other larger trunks. The arteries are mostly found with greatly thickened walls and when we remember the special anatomy of the pancreatic vessels, it is ob-

vious that changes are more commonly found in the intima than in the adventitia, which in these trunks is usually scant. Generally stated, calcareous infiltration has been found rather less frequently in the pancreatic vessels than in the visceral arteries and even when general arterial calcification is present, the pancreatic vessels show the change less pronounced or not at all. Contrarywise fatty degeneration and atheromatous cysts are found more commonly. The lesions in nearly all cases are most evident in the intima with secondary or alterations of lesser degree in the media. In no instance has actual aneurysmal dilatation been demonstrable, though in a great many cases the vessel has been dilated as a whole or for considerable distances.

The frequency of arterio-sclerosis in the trunks of the pancreas may in part be explained by the anatomical arrangement. In general they are of considerable size and length and their course lies mostly in a soft and unresisting tissue which supports them but very insecurely. We should, therefore, expect, for example, that when the blood-pressure was raised, the pancreatic trunks would suffer more than most other vessels on account of this lack of firm external support. This factor probably accounts for the greater relative involvement of the arteria pancreation magna. The vessels of the pancreas are also without exception branches of main arterial trunks which are in themselves prone to arterio-sclerosis, as the splenic, mesenteric and hepatic arteries. The blood supply to the pancreas is very abundant and at the same time it is one which at certain periods, as during digestion, must be very actively increased, and during resting stages the circulating blood must be curtailed by contraction of the media coat. In order to meet with these primary essential requirements these vessels must then have a well developed media or muscle coat and throughout the visceral arteries it is notable that those which possess activity of function to a more marked degree are those most affected in visceral arterio-sclerosis.

Alterations in the Pancreatic Tissue—Changes in the pancreatic tissues of a commensurate grade have been found in every instance of marked arterio-sclerosis. These lesions may be mostly grouped under two heads. The first, which comprises the more frequent cases, are those in which fibrosis of the pancreas is present. This differs greatly in degree but is found to vary quite constantly with an apparent definite relationship to the severity of the vascular lesions. Thus, when the fibrosis is found very marked, the arterio-sclerosis is extreme and vice versa. It must also be pointed out that, for the greater part, this fibrosis is found, not as a special manifestation of a general process, but as a distinctly local one and therefore apparently more or less definitely associated with the visceral arterial disease.

Both interlobular and interacinar fibrosis is found, the former rather more frequently in less marked instances. In many cases the fibrosis is of so extreme a degree as to cause extensive parenchymatous pressure atrophy and when the ducts are compressed, cyst formation develops. In practically all the diffuse cases, when atrophy of the parenchyma is present, both the ordinary acini and the bodies of Langerhans suffer in apparently equal degree, and this appears to be regularly true when either the so-called interlobular or interacinar fibrosis is present. In the latter case, of course, parenchymatous atrophy is more marked as a rule, though in many the interaciner process appears to be but a later stage of the interlobular form.

The other type of pancreatic disease commonly associated with local arterio-sclerosis is fatty infiltration. It is in the majority of cases also found with parenchymatous atrophy, and is, of course, in some, but by no means in all instances, concurrent with general adiposis. In the greater number of cases this fatty infiltration consists of a simple over-deposition of adipose in the stroma of the gland and the resulting parenchymatous changes are in the nature of a pressure atrophy. Fibrosis of greater or less degree is often associated with this lesion and in some cases these changes result in very extensive parenchymatous replacement.

Arterio-sclerosis was present in four cases of fat necrosis. In two of these no other etiological lesion could be made out and the arterial disease seemed to be possibly connected in a direct causative way. In the remaining two cases the lesion could be more definitely ascribed to leakage of pancreatic secretion following ulceration and the lodgment of a stone in the pancreatic duct with fibrosis and cyst formation. The relationship which arterial disease bears to fat necrosis is still a mooted point and space does not permit its discussion here, suffice it to say that it appears to me that in small and isolated foci the change may be due to an arterio-capillary fibrosis associated with a cutting off of the nourishment of minute nodules of fat. Of course in the majority of cases, and I believe in all instances in which the lesion is extensive, it is due to the escape of pancreatic secretion, often mixed with extravasated bile.

Pancreatic arterio-sclerosis apparently bears no relationship to haemorrhagic pancreatitis and the three instances of this disease which have occurred in my service have been found independent of arterial changes.

Relation of Pancreatic Arterial Disease to Diabetes Mellitus—In the 75 cases which are the basis of this study, glycosuria appeared as a symptom in but 8. Three of these were in cases of acromegalia in which glycosuria is probably a symptom independent of diabetes mellitus in its pure type. The association of the lesions of arterial disease with diabetes mellitus can, therefore, be asserted in but 5 out of the 10 cases of diabetes melitus which ap-

pear in my autopsy records. It is important to note in this particular that all 10 showed more or less fibrosis of the gland, 5 were, however, relatively free from arterial lesions. On the other hand 10 cases of extreme fibrosis or sclerotic pancreatitis with profound parenchymatous atrophy were unaccompanied by glycosuria. In the present state of our knowledge the exact relationship, either between pancreatic fibrosis and diabetes or arterial disease and pancreatic diabetes admits of no definite arrangement, for in several of my 10 cases of extreme fibrosis without glycosuria, extreme atrophy and necrosis of the bodies of Langerhans was shown. As matters now stand the ingenious theory of Opie can be neither disproven nor substantiated on an anatomical basis.

Symptomatology—Like the symptoms of all lesions of the pancreas, those which follow arterio-sclerosis with its attendant alterations in the organ are, even in the most pronounced instances, usually irregular and indefinte. Symptoms which might be justly attributed to disorder of the pancreas are present in a little over half of my cases. Several were, however, studied before there was so general a familiarity with pancreatic disease. In some cases in which I feel certain symptoms must have been exhibited, no sufficient inquiry as to their existence appears to have been made.

Symptoms of digestive disturbance are found to be the most constant of any in my series. These vary from simple nausea, flatulence and intestinal fermentation with occasional attacks of diarrhoea, to severe vomiting, persistant nausea and constipation or active diarrhoea, in the more pronounced examples. In some, in which the character of the vomitus or stomach washing was studied, bile was ordinarily present or the gastric fluids were found natural. Perhaps these symptoms may be best summarized as being those of a dyspepsia chiefly manifest when foods rich in albumens or fats were taken, but without the chemical evidences of true gastric dyspepsia.

Sialorrhoea, a symptom stated by several writers to be common in pancreatic lesions of a destructive character was found in but a single one of my cases. In this instance a large sized cyst of the pancreas was present associated with extreme local arterial disease and pancreatic sclerosis. The symptom was very marked in this case and was complained of most bitterly by the patient. This unusual symptom is very apt to pass unnoticed unless it is sought for, even in cases where it is well marked.

Thirst of marked degree was present in a few cases, again in only the more pronounced ones. This symptom has of course been entirely disregarded in instances showing glycosuria where its explanation would be much more simple than the theory accounting for it in pancreatic disease without glycosuria.

Jaundice of slight degree has been noted in a few instances. It was quite marked in the cases in which fat necrosis was a complicating factor.

In one of these it must be noted, however, that partial obstruction of the common duct from a gallstone was a probable factor in the production of the symptom. Jaundice is said by Keene to result in some instances from compression of the common duct by fibrous hyperplasia in sclerosis of the pancreatic head. In many cases it has been apparently due to inflammatory conditions of the duct either excited by the presence of calculi, or from probable infection. In one instance in which this symptom was marked, the mucosa of the duct of Wirsung was extensively ulcerated.

Pain, localized in the pancreatic region, has been one of the most frequent symptoms in the cases under my clinical observation. It has been mostly described as of a dull, gnawing character, and has usually excited suspicion as to the presence of gastric carcinoma. Associated with the pain in most cases is a spot of tenderness with distinct localization in the area of the pancreas. In several, especially in two patients recently under my observation in which the arterial changes were found post mortem to be very pronounced, this pain took on a decidedly anginal character, was paroxysmal, very severe and was accompanied with paleness, faintness and in one case with syncope. This last case finally terminated with acute fat necrosis and death. In this instance anginal attacks affecting the coronary and anterior tibial vessels were also seen. Keene states that the pain generally radiates toward the left side of the abdomen. I have not observed this, but it seems logical to suppose that it does, assuming it to be due either to arterial or to duct spasm.

The tendency to general as well as to local haemorrhages noted by some, especially by Mayo Robson as present in cases of destructive lesions of the pancreas has not appeared in any which have fallen under my observation, nor have I found the tendency obvious in any of the more actively destructive lesions of the pancreas, as acute necrosis or new growths, some ten cases of which I have on record. The haemorrhage in pancreatitis haemorrhagica, as conclusively demonstrated by Flexner, admits of a much more definite explanation.

The character of the stools has been disappointing from a diagnostic standpoint in my cases, most of which showed nothing of this nature which could excite suspicion. In a few steatorrhoea has been noted at times, but its degree was not sufficient to justify much weight being laid upon it. Azzootorhoea has been reported in a few cases of more pronounced character, but this sign is at best uncertain and I have found it very difficult to satisfactorily determine. This is so because the amount of muscle varies so greatly in the stools of normal individuals under different dietetic and physiological conditions. The volume of the faeces has not been notably great in any of my cases and excessive elimination of muscle has been probably significant in but one instance.

Wasting of the tissues, quite independent of the association with diabetes, has been present in a considerable number of cases, enough so to make it appear that this is quite a constant symptom of pronounced arterio-sclerosis and its attendant changes in the pancreas, as it is in most other destructive lesions of this organ. In a few instances, instead of emaciation, adiposis associated with marked anaemia, almost cachectic in type, has developed.

Changes in the urine, except for the occasional occurrence of glycosuria, have not been noted in any of my cases, most of which came to me before I was familiar with the Cammige reaction which now appears to promise data of great diagnostic value in destructive lesions of the pancreas. Neither has the neuleinic test been tried, except in one case in which negative results followed.

Attempts at the experimental production of arterio-sclerosis with fibrosis of the pancreas by the administration of vaso constrictors as ergot and adrenaline; of vasodilators as the nitrites and chloral have been entirely negative. Neither did the condition follow in experiments on rabbits by the introduction of alimentary abnormalities, as in the use of large amounts of animal and vegetable fats, putrid meats, alcohol in various forms, of lead salts and large amounts of irritating condiments though used sufficiently to set up a gastro-duodenal catarrh of chronic nature. It now appears to me that we have absolutely no experimental evidence on which to base our theories as to the probable factors at work in the formation of arterio-sclerosis of the pancreatic vessels. But the fact remains that the condition must be recognized by the clinitian and considered in the differential diagnosis of all probable destructive pancreatic lesions.

THE CHOICE OF DIURETICS IN CARDIO-RENAL DISEASE.

By C. E. Quimby, M. D.

Attending Physician.

The difficulty and uncertainty in the choice of a diuretic, which so frequently oppress the young men as they assume the responsibilities of a medical service, are the excuse for this paper, which makes no pretense of being exhaustive. It is designed simply to present in elemental form the general principles upon which any logical selection of diuretics must be based, and to afford assistance to successive house officers in solving those therapeutic problems that are so constantly presented in the medical service of this hospital. With young men entering a hospital service, pathology, with its inevitable idea of a dynamic perversion, dominates their mental processes, largely to the exclusion of physiology, so that in their consideration of disease they think almost entirely of results rarely of processes; they think from the autopsy backward, not from the inception forward. Their mental pictures never portray disease as the harmonious activity of normal physical forces, directed to the elimination, never of themselves, but some extraneous force; or, as is more frequently the case in this hospital, simply to the restoration of functional competency. Yet without such a picture the treatment of cardio-renal disease must be empirical, uncertain and shifting with every new product of the synthetic-materia-medica fanatic.

The primary requisite, for an intelligent use of diuretics is recognition of the fact that urinary secretion is a physiological process, determined by functional power with a maximum limit, and dependent upon a mechanism which is not single, but compound, and in which failure of any single component is equivalent to failure of the entire mechanism. Upon this basis diuretics at once separate into two general classes: 1st. Those decreasing work to be done or functional demands; and 2d. Those increasing functional activity or power. Logically the first class includes several of the drugs, but clinical convenience pardons its limitation to diet and the use of fluids. The necessity for modification and restriction of diet is universally recognized and applied whenever there is gross disease of the kidney or extreme lack of functional activity; but the frequency of moderate, persistent excess in demand for renal activity as the etical factor in definite renal disease, and the value of this diuretic, carried even to the point of absolute starvation, in some acute conditions, are by no means as generally appreciated.

Chronic Bright's disease begins, in the vast majority of cases, as a general perversion of metabolism, excited by either simple strain or toxic inhibition of function, and continues for years presenting just as definite signs of renal distress as are suppression or coma. It is in these early stages of

functional strain precedent to organic injury, which are too frequently ignored, that Bright's disease is definitely curable. In this stage the obtrusive symptom may be anything from grumpiness to goitre, from pestering activity to angina pectoris, but the two earliest, constant and most significant are accentuation of the aortic second sound and persistent disturbance of the urinary secretion, either in quantity or ratio between solids and fluid. Under these conditions modification of diet is the one imperative diuretic without which all others prove useless. But with an appropriate diet and the persistent use of the common alteratives, as arsenic and mercury, not only will the aortic sound and urine regain their normal characters, but the distressing, and apparently unrelated subjective or objective symptoms will also disappear as well.

It is not in chronic conditions alone that diet is our most powerful diuretic. Not infrequently a mitral leak of long standing will be ignored by a robust patient until the heart, under prolonged abuse, lets up rather suddenly on the right side, with the coincident development of anasarca, renal failure and scanty albuminous urine. Under these conditions, with a regular, high tension pulse and the left heart sounds strong and clear, there is no diuretic whose action is more speedy, satisfactory or permanent than absolute starvation for varying periods up to a week. Case J. C. was found sitting up in bed gasping for breath, oedematous to the arm-pits; urine scanty, with moderate amount albumin, heart rapid, but regular, and sounds strong and clear; distinct old mitral leak. Patient had been previously growing worse for past week on full doses of digitalis. Under practically absolute withdrawal of food and fluids, patient was able to sleep lying prone within thirty-six hours and was out driving in two weeks, and lived several years thereafter. The indications for this diuretic in acute conditions are, therefore, strong vascular activity with diminished renal secretion, dependent primarily upon back pressure in the veins.

In the use of the functional diuretics our choice is based on the principle already stated, that the urinary secretion is dependent upon a composite mechanism in which failure of one part means failure of the whole. In view of the fact that not only do different parts of this mechanism fail in different cases, but that other parts frequently attempt to compensate for such failure with injurious results, it is important to examine this mechanism and make clear the relations of its several parts and their reactions one upon the other. It is composed of nerve centres and vaso-motor nerves, of the heart, the arteries (and the two are separated because they act separately), of the renal circulation, and the secreting cells. For each of these components, representing an independent function, we have stimulant and sedative diuretics. The first three work together to control and regulate the circulation through the kidney in proportion to the absolute amount of

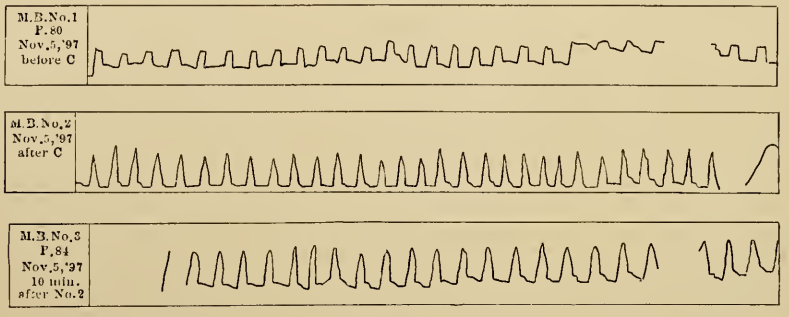
work to be done and the functional power of the kidney. Moreover, increased work and decreased function excite the same reflex activity in this vascular mechanism. It is evident that our choice of vascular diuretics will depend upon our belief as to the element in the circulation determining functional activity. Since increase of vascular tension is nature's only measure for augmenting circulation, and since such increase of tension does normally result in increase of function, it was and is easy for the physiologist to assume that blood pressure is the sole determining factor of functional activity. Although trained in the strictest form of this belief, personal experiments undertaken some fifteen years since to determine the effect of mechanical control of the circulation under low blood pressure convinced me beyond the possibility of a doubt that the quantity of blood passing in a given time, and not pressure, is the modifying factor in function, so far as concerns the circulation; and that blood pressure above a certain point, especially in organs with a firm envelope, actually inhibits function. Upon this basis we shall consider some of the diuretics applicable to the several parts of the vascular mechanism.

Primary failure of the nerve centres is exceedingly rare, and when developed acutely is usually from cerebral pressure, as indicated by Cheyne-Stokes breathing, and requires mechanical rather than drug treatment. When it is part of general systemic exhaustion it often happens that the addition of strychnia to diuretics already in use will result in the desired diuresis. In milder conditions of the same character when all the symptoms, and especially those from the vascular system, indicate "weariness," caffeine will be found the more satisfactory, supplying both the nerve stimulant and the direct diuretic. Conditions of over-activity in the nerve centres are, on the other hand, only too frequent. Why, in one man the nerve centres accept a certain amount of toxaemia composedly, while in another they set the whole system in a tumult is inexplicable. The fact, however, finds constant illustration in cardio-renal disease. This condition is indicated by nervous symptoms and vascular activity out of all proportion to the degree of renal inadequacy. The patient is often exicted, the face flushed, the heart pounding with sharp valvular sounds, the pulse full and hard, while the urine, despite the high tension, is diminished. Remembering that the urine varies with quantity, not pressure of the blood, it is easy to see that in this condition excessive reaction has raised the blood pressure to the point at which, by tissue compression, it inhibits instead of stimulating renal activity. For its relief and the restoration of urinary secretion aconite is often the most satisfactory diuretic, remembering only that its action is simply to quiet vascular activity and lower blood pressure, which, although frequently sufficient of itself to restore renal function, may at times be aided by more direct diuretics. That codeine has a somewhat similar action must be men-

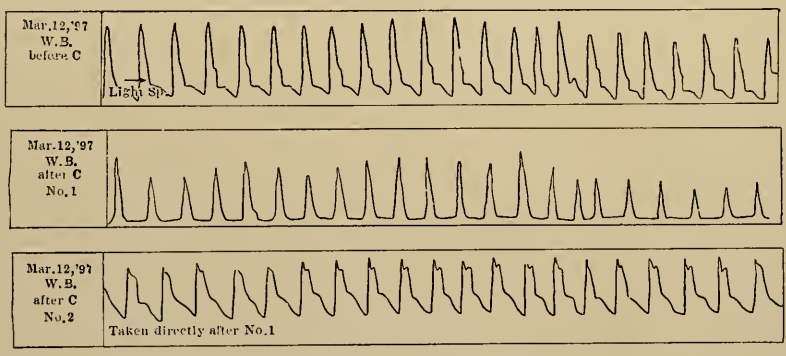
tioned, though space forbids comparison in detail. In general, codeine or morphia is for temporary use in the very acute conditions, and in mild chronic cases when we wish also to diminish work by inhibiting metabolism.

Although the heart and arteries work together to a common end and with a delicately adjusted complemental action, this compensatory action itself often augments the very condition it is designed to remove, for the heart and arteries do not always hold out equally under strain or respond proportionately to vascular stimulants. Given the prolonged strain of cardio-renal disease, not only is it impossible to say whether the heart or arteries, and in the heart itself, the right or left side will be the first to give way, but with failure present, it will often shift from time to time without apparent cause from one to the other. The action of the vascular diuretics will be good or bad according as they are adjusted to these varying conditions in each case. The pertinency of the foregoing finds constant illustration in the use of digitalis, which is the type of the cardio-vascular stimulants. Since digitalis is diuretic by reason of its influence on blood pressure, the first and most important rule is, " do not use digitalis with definitely high blood pressure and strong heart action, especially if the pulse is large and full, letting up but little during diastole," which indicates beginning weakening of arterial as compared with cardiac power. The exceptions to this rule are acute conditions in relatively young subjects whose muscles can be trusted to endure sharp stimulation. With definite vascular exhaustion and low blood pressure digitalis is either our most valuable or most dangerous diuretic. Broadly speaking, its value is in direct proportion to the absence of degenerative changes in the cardiac muscle. It is, therefore, more certain in its action the younger the patient. It may be considered safe and the appropriate diuretic so long as the heart sounds maintain their normal ratio of intensity; but with older patients, certainly after fifty, and when, with relatively weak heart sounds, the second is distinctly accentuated, indicating a tendency to cardiac incompetence, the use of digitalis should be tentative at first and begun with small doses, since its action on the arteries precedes that on the heart. By a very cautious and gradual increase of dosage it is sometimes possible to secure the desired effect in this border-land class of cases where its rapid administration in full doses will only provoke the threatened cardiac failure and urinary suppression. The failures of digitalis in this class of cases and its absolutely harmful action in the stages of their later development are due to its causing arterial contraction out of all proportion to its influence on the heart. These border-land cases are at the point at which harmonious action of heart and arteries is failing and the independent compensatory action begins. That these conditions of cardiac " let up " with increased arterial contraction, which is plainly compensatory for the lessened heart push, are actual can not be doubted by one who has

studied a series of pulse tracings taken at frequent intervals. The following two sets of tracings not only afford sufficient proof of that fact, but also demonstrate how moderate a relief may enable an exhausted heart to recover its power and thus make plain the rationale of the proper diuretic for these conditions. In the first set, from a cardio-renal case, the first tracing shows the small pulse and prolonged tension of arterial contraction with weak heart action. The second shows the effect of arterio-capillary dilata-



tion in lowered blood pressure, and the third, by comparison with the first, measures the gain in heart power resulting from twenty minutes' rest under such forced dilatation. Of the second set, from a cardiac case, the first shows both cardiac and arterial weakening, the second gives the tension ratios under complete capillary dilatation, and the third again marks the



restoration of both cardiac and arterial power from a similar twenty minutes' rest. In each case the third tracing was taken within ten minutes after the second.

These relations make it easy to understand how, under the strain of prolonged high tension physiologically developed for the maintenance of renal function, the heart gradually fails and the blood flow is not only lessened, but a back pressure developed, even when there is no defect in the valves.

The direct reflex from the consequent deficient blood supply is still more arterial contraction and increase of blood pressure which, although physiologically compensatory, in fact still further impedes the heart and thus increases the condition it is designed to relieve. If the right heart holds out, the earlier stages of this process may be suggested only by slight but easily increased dyspnoea. More commonly the development is insidious and unnoticed until the venous tension amounts to such compression of the kidney as, joined with the diminished blood flow, causes serious failure of function. It is in these conditions, characterized by deficient urine, a small, firmly contracted artery and weak pulse, weak heart action and sounds, and an absolutely low blood pressure, with or without oedema, conditions that are so constantly assumed to demand the use of digitalis, that digitalis is rank poison, while glonoin, either alone or in combination with codeine, is a strikingly effective diuretic. The rapid increase of urine under its action proves that the amount of blood passing through the kidney is increased by the relief of the venous compression, although the larger proportion of the blood is drained into and retained in the dilated arterial system. These results are not secured by 1/100 of glonoin three times aday, but by 1/50 or more every fifteen minutes until definite results are manifest in the circulation, and then every hour or two for a day or a week, as may be required. Of course, such dilatation of the arteries can not be made permanent, and the time comes when the heart must take up its work. Its ability to do this may be tested by simple withdrawal of the nitroglycerine. If the result is satisfactory, its power may be further tried by the cautious use of small doses of digitalis. It will often be found that a heart previously utterly intolerant responds quickly and perfectly to digitalis thus used after a prolonged rest under glonoin. Once under control it may usually be held for a long time, but the digitalis were best never entirely omitted and the dosage should be reduced only under careful oversight. When such a heart "gets away" the second time it is regained with much greater difficulty.

Of the direct diuretics it is unnecessary to consider the salines beyond calling attention to the fact that they are not the only diuretics, as young men are so apt to think, and saying that they are seldom indicated in cardio-renal disease. For the others calomel and squill will serve to illustrate their use. Mercury is the most valuable stimulant for the renal epithelium which we possess. It is both a direct and indirect diuretic, and is indicated in two very different sets of conditions. First, when the effects of the venous tension following heart failure are particularly manifest in the liver and intestines, calomel given to the point of profuse catharsis is a diuretic not only through the relief of venous tension, but later by its direct action on the kidney. When the case has reached the point of intestinal paresis, with tympanitis and oedema, the action of the calomel is made more certain by

the addition of aloes. As the diuretic action follows the catharsis this later should be inhibited by opium after the first thorough clearing of the bowel. Usually one or two grains of calomel every two hours for ten doses will suffice if opium is given with the last five. It may, however, be continued longer if necessary, but should be stopped as soon as the diuretic effect is well established, as the diuresis will usually continue until normal conditions are regained. The other condition indicating calomel is that of plain failure of renal epithelium. The distinctive symptom of these cases is excessive urine with low specific gravity. In their earlier stages they are the ones for which diet was prescribed. At that time no sudden change in the urine is to be expected, but the persistent use of small doses of mercury even then will be followed by such changes in specific gravity as demonstrate a diuretic action. In the later stages when more advanced renal changes are shown by abundant albumen and casts, or renal failure has even caused severe oedema, the amount of urine being still in excess, there is no diuretic which equals calomel. It is to be used very differently, however, from before. There is no better combination than the historic "Guy's" pill, the grain each of calomel, squill and digitalis for the general run of cases, and, as a rule, three or four a day are sufficient. But these cases are very close to those in which heart failure and back pressure are involved. If, then, the heart is doing all right, as shown by the abundance of the urine, it is often wise to leave out the digitalis until the diuretic action is established; and if, by any chance, the quantity of urine diminishes under the use of the full pill without corresponding increase of specific gravity, then the digitalis is certainly doing harm. Salivation from this pill may be avoided in part by use of a mouth wash, or the bichloride of mercury may be substituted for the calomel when its use must be long continued. Certain writers have recently seen fit to condemn the use of calomel in renal disease. Fortunately, however, nature is yet unacquainted with the fact, and calomel still continues to exercise its beautiful diuretic power as it has for half a century or more.

AN ACUTE CASE OF HODGKIN'S DISEASE.

Wm. R. Williams, M. D.

Assistant Attending Physician.

Last summer a patient was admitted to the City Hospital and the following history, with the help of an interpreter, was obtained.

Male, Italian, 72 years old, a laborer, admitted August 28, 1907.

Family History.—Irrelevant.

Personal History.—Drinks moderately of alcohol. Contracted syphilis fifty years ago. The secondary symptoms lasted two months. Married forty years ago and had three children, of whom two died in infancy; the third is alive and well. Has always worked hard. "Fever" fifteen years ago. For several months has been troubled by dyspnoea on exertion.

Present Illness.—A month ago he noticed some lumps in his neck, soon followed by swellings in the groins and axillae. He lost strength rapidly and on August 28 was admitted to the City Hospital complaining of the swelling of the lymph nodes and of weakness.

Physical Examination.—A spare man, six feet tall, weighing 131 lbs. The skin of the face is somewhat bronzed, that of the rest of the body is pale and flabby. The muscles are small, soft, and toneless.

Cerebration seems normal. The pupils react normally to light and accommodation. Conjunctivae are pale. The ears and hearing are normal.

The tongue is protruded mesially and shows a fine tremor. It is large, thick and coated. Pyorrhoea alveolaris is present.

Thorax.—The chest is somewhat barrel-shaped, the lower border of the ribs is prominent and there is a chain of dilated capillaries along the left costal margin. The skin of the back shows scars from the thermo-cautery. Expansion is only fair. Respiration is regular, the rate is 34 to the minute. Vocal fremitus is distinct and the resonance somewhat exaggerated over both lungs everywhere. The breath and voice sounds are normal. No rales are heard.

Heart.—The apex impulse is not seen or felt. Epigastric pulsation is present. The left border of cardiac dullness is within the nipple line, the right border is indistinct. The apex, as determined by auscultation, is in the fifth space within the nipple line. The sounds at the apex are of poor quality, there is no murmur. The aortic second sound is sharp and somewhat accentuated. The arteries are thickened and tortuous. The pulse is regular, 128 to the minute. The blood pressure is 150 mm. of mercury.

The liver dullness extends from the sixth space to the free border of the ribs in the midclavicular line. The edge is not felt. The spleen is not distinctly felt, but there is tenderness at the margin of the ribs on deep inspiration.

Rectal examination is negative.

The knee jerks and plantar reflexes are normal.

The lymph nodes are very generally enlarged. They form chains on both sides of the neck, there are masses in the axillae, groins, Scarpa's triangles, and popliteal spaces. The nodes are somewhat movable on the deeper structures, the skin over them is not adherent. They are firm and do not fluctuate. The largest nodes are about four centimetres in diameter. Those in the right groin are slightly tender. The nodes in the posterior

triangles of the neck, the epitrochlear nodes, a node lateral to the left nipple and one in the right midaxillary line opposite the fifth rib are enlarged. The abdominal nodes are not felt. Signs of pressure within the thorax are absent.

During the patient's stay in the hospital he was kept in bed. His temperature ran an irregular course, the daily maximum ranging from 100° to 102° F., the minimum from 98° to 99° F. He had no chills or sweating.

Blood Examination, August 28—Red cells, 3,528,000; leucocytes, 14,000; differential count, polymorphonuclears, 70 per cent.; lymphocytes, 25 per cent.; large mononuclears and transitionals, 4 per cent.; basophiles, 1 per cent. Two megaloblasts were seen while counting 300 leucocytes.

September 5—Red cells, 3,750,000; leucocytes, 8,000; polymorphonuclears, 66 per cent.; lymphocytes, 30 per cent.; large mononuclears and transitionals, 3 per cent.; basophiles, 0.5 per cent.; eosinophiles, 0.5 per cent. Nine normoblasts.

September 10—Red cells, 4,136,000; whites, 12,000; polymorphonuclears, 70 per cent.; lymphocytes, 20 per cent.; transitionals, 7 per cent.; eosinophiles, 2 per cent.; basophiles, 1 per cent. Seven megaloblasts and four normoblasts.

Urine on admission—Acid, Sp. Gr. 1020, trace of albumin, no sugar, a few hyaline casts.

The patient steadily lost strength. A few days before death there were rales over the back of the chest. He died on September 14 with oedema of the lungs, after an illness that lasted less than two months.

Autopsy by Dr. Martland, twenty hours post mortem. The protocol, with the omission of the description of the superficial nodes, may be summarized as follows:

On section there is a moderate amount of yellow moist fat. The muscle is reddish brown, and in good condition. Almost all of the glands on the side of the chest are enlarged, especially over the ribs, where they are soft and white, and when they are scraped away, the periosteum looks rough and worm-eaten. The diaphragm is at the level of the fifth space on the right and the sixth space on the left. The liver presents 10 cm. below the ribs in the right midclavicular line, and 9 cm. below the xiphoid. It is brownish red and contains whitish nodules that are slightly elevated. Just above the lesser curvature of the stomach, in the gastro-hepatic omentum, are a few small, white, nodular glands. A larger gland 2 cm. in diameter is seen above the pylorus on the portal vessels.

Thorax—The ribs are somewhat calcified. The glands in the anterior mediastinum are enlarged, white, soft, and adherent to the sternum. The left pleural cavity is obliterated by adhesions at its anterior border. The cavity contains some serum. The right pleura is free and contains some

serum. The lungs are emphysematous and oedematous. At the apex of the left lung are old puckered scars which do not extend deeply into the lung substance. This lung was closely adherent to the chest wall along its posterior and anterior borders and to the diaphragm. The peribronchial glands are anthracotic and contain small, softer, white areas.

The pericardium contained 90 cc. of clear straw-colored serum.

The heart weighs 370 gm. Between the aorta and the pulmonary artery are two small soft glands. The tricuspid orifice admits three fingers, the valve contains some small gelatinous nodules at its base and at the line of closure. The pulmonary valves and the pulmonary artery are normal. The mitral valve is normal except for some irregular patches just beneath the aortic valve. The orifice admits two fingers. The aortic valves are somewhat stiffened at the base. The aorta throughout, but especially near the orifices of the coronaries, contains numerous yellowish grayish patches. The heart muscle on section is pale brown, the muscle bundles are not distinct. The coronaries are rather large, have thick walls and show yellow patches. The endocardium of both ventricles shows some white patches and streaks. The spleen is rather large, soft and pulpy. The upper half was adherent and was torn in removal. Its size is 16 by 10 by 5 cm. The capsule is white and thickened in patches. The cut surface is studded with large and small soft white nodules of an average diameter of 2 mm. These project above the surface and are surrounded by reddish spleen parenchyma. They seem to be hyperplastic Malpighian follicles.

The suprarenals are large, the cortex thick, the medulla in places white and soft.

Each kidney weighs 190 gm. The capsule strips easily, leaving a finely granular surface. The cortex has an average thickness of 5 mm., and is pale. The glomeruli stand out as distinct white glistening points. In the kidney substance are three white nodules, 3 mm. in diameter.

At the base of the tongue are a few raised whitish nodules at the site of the follicles.

The larynx shows a few white follicles in the aryteno-epiglottic fossae. The deep lymphatics of the neck and thorax are enlarged and press closely on the trachea, but do not obstruct its lumen.

The liver is rather large, firm and brownish red. The capsule is marked by a few whitish streaks. Just beneath the capsule in the left lobe are a few yellowish white, slightly elevated nodules, which on section, extend into the liver substance from 5 to 10 mm. They are soft and the liver parenchyma around them seems normal. Similar masses from 5 to 15 mm. in diameter appear throughout the liver. The region of the intralobular veins is distinct as a dark red area and it is surrounded by a light yellowish peripheral zone.

The glands around the iliac vessels, continuous with the glands of the groin, the retroperitoneal glands and the thoracic glands along the spine are all enlarged. The lymphatic duct seems to be free, but on cutting the receptaculum chyli a whitish, pulpy fluid escapes. Sections of the marrow from the ribs and the middle of the femur show pale, oily marrow, with extensive lymphoid involvement. The lymphatic tissue shows the microscopic structure described as characteristic of Hodgkin's disease, the endothelial hyperplasia, the multinucleated and giant cells, and the eosinophile cells. The fibrous connective tissue is very scanty. An emulsion of the nodes was injected into a guinea pig. He did not develop tuberculosis.

Although Malpighi described a condition of general lymphatic enlargement with nodules in the spleen as long ago as 1669, it was not until Hodgkin, an English Quaker, published his account of eight cases in 1832, that the condition was recognized as a distinct disease. Since the appearance of this paper interest in the condition has not flagged. A very large number of cases have been studied and the diagnostic criteria have been defined and re-defined with increasing precision. Many of the cases heretofore reported as examples of pseudoleukaemia would not now be recognized as such, but would be classified with tuberculosis, syphilis, lymphatic leukaemia, or lymphosarcoma. Indeeed, it is claimed that of Hodgkin's cases, one was tuberculous and another syphilitic. Although the endothelial hyperplasia and the giant cells were described long previously, the recent work of Dorothy Reed seems to have crystallized our knowledge of the histology of the tumors in the lymphoid structures. By a study of the blood and of sections of the nodes we may now reach a satisfactory diagnosis in most cases, although the pathogenesis of the disease is as obscure as ever. A number of cases are on record where the blood picture has changed in a most striking manner from that of a secondary anaemia such as is found in Hodgkin's disease to that of a typical lymphatic leukaemia. The classification of this group of cases is not as yet satisfactory.

The shortest duration of symptoms in the cases reported by Hodgkin was "three or four months." This was in a patient whom he did not see, but who was observed in Paris by his friend, Dr. Carswell, who gave him the clinical and autopsy notes. Since that date several cases have been reported where the course was as short as this or even shorter. Some of these, however, seem more properly to be regarded as acute lymphatic leukaemia. Thus Dreschfeld, in 1891 and 1892, reports as Hodgkin's disease a case that lasted only six weeks, of which he says that the white blood cells were mostly small mononuclear forms and that there was one white cell to about forty reds. Another of his cases had a ratio of 1 to 30 between the whites and reds, and here, also, most of the leucocytes were small mononuclears. A third of his cases of acute Hodgkin's disease lasted seven

weeks. Here, also, he said that the white cells were mostly mononuclears. Their ratio to the reds was 1 to 100, but as the reds numbered only 2,000,000, and he does not give the exact percentage of lymphocytes, it is possible that this case would now be called Hodgkin's. In Gower's very careful article in Reynold's System (1880), he gives the duration of this disease as "three or four months to several years." Within recent years, however, a few cases have been reported in which repeated blood examinations excluded leukaemia and in which the duration of symptoms was from five weeks to three months.

The case reported here, therefore, is among the most rapid, and, beside the blood picture, the histological examination of the tumors is offered as a decisive point in the diagnosis. It is of interest, also, because of the very general implication of the lymphoid structures of the body; the follicles of the intestines being almost the only foci to escape. There were nodules in the liver, kidneys, at the base of the tongue and in the marrow, and there was serum in the serous sacs of the thorax. None of the nodes was particularly large, the greatest being four centimeters in diameter.

There was an old focus of tuberculosis in the left lung that apparently had long been perfectly healed. No history of any symptoms due to this was elicited. There was a history of syphilis, also, but there had been no symptoms for half a century.

Throughout his stay in the hospital the patient ran a very irregular temperature that did not suggest the curve described as recurrent glandular fever with its rhythmic alternation of periods of pyrexia and of normal temperature. The blood examinations did not at any time show a high grade of anaemia, nor was there any suggestion of a change to the picture of leukaemia.

As to the treatment of this case, there is nothing to be said. That it was unavailing is obvious. Fowler's solution was given in increasing doses. Although there was a history of dyspnoea on exertion several months previously, there seemed to be no indication for special treatment directed to the heart, arteries or kidney.

NON-FATAL COMA IN THE COURSE OF DIABETES.[1] [2]

By C. N. B. Camac, M. D.,

Attending Physician.

The case of diabetes here reported was observed clinically for a period of nine months. The patient developed coma, from which she recovered, and four months later died in hemiplegia. Postmortem examination was made. The opportunity to follow such a sequence of events being comparatively uncommon, the report is thought to be of sufficient interest to add to the records now accumulating with regard to coma in diabetes as well as coma in general.

Report of the Case up to the Development of the Coma (In Brief).—A woman, aged forty-eight years, was admitted to the City Hospital, February 27, 1907. Her complaint was nausea. Syphilis had been contracted, but when was doubtful. The patient was of feeble mentality; talkative. There was considerable thirst, large appetite, and other symptoms of diabetes, which seemed to have existed for about two years. There was a moderate degree of general arteriosclerosis. Chart I presents the urinary findings up to the time the coma developed.

Report of the Coma.—For the first two and one-half months after the patient was admitted a detail examination of the urine was recorded (Chart I). This was then discontinued until June 3, when the house physician reported that the patient had recently become irritable, had refused to save the urine, and had considerable diarrhœa, which had resisted ordinary remedies. For several days past (June 3) she had complained of weakness and muscular pain, especially in the back. She frequently fell asleep in her chair. There was some incontinence of urine and feces (diarrhœa) at these times. On the day previous (June 2) she had slept in her chair the greater part of the day, but would arouse as persons approached, and remain awake while they conversed with her. On June 4 the drowsiness markedly increased; she responded only when shaken, and then complained in a heavy manner of general pain, and quickly lapsed into her former conditions. There was incontinence of urine and feces. The breathing was audible, like that of one in deep sleep. It was never labored or irregular, that is, no dyspnœa (Kussmaul) was observed. June 5 the patient could not be aroused. Alkaline treatment was instituted on this date: Milk diet; bicarbonate of sodium, xxx grains (2 grams) by mouth every hour;

[1] Read at a meeting of the Association of American Physicians, Washington, D. C., May 12 and 13, 1907, and published in The Transactions of the Association, Vol. XXIII.

[2] Extracted from the American Journal of the Medical Sciences, December, 1908.

saline enemas, one pint (500 c.c.) every six hours; hypodermoclysis (saline), one pint (500 c.c.) twice daily. If the urine remained acid to litmus or the patient's condition did not improve, bicarbonate of sodium was to be administered subcutaneously and intravenously. If an alkaline reaction of the urine appeared, the quantity of bicarbonate of sodium was to be reduced. Chart II presents the urinary findings during the coma.

It will be observed in this table that during the coma the specific gravity fell; the volume for twenty-four hours was about the same as that before the coma; the reaction was acid until June 8; the sugar was much reduced; acetone and diacetic acid were present in small quantity and β-oxybutyric acid was absent; ammonia was low; indican was not increased and there was a moderate amount of albumin and occasional casts. With the clearing of the coma the diacetic acid and acetone disappeared (in this order), the amount of sugar and the specific gravity increased. The urine remained acid to phenolphthalein, but on June 8 was alkaline to litmus. This, together with the lessening of the coma, contra-indicated an increase in the amount of alkali to be administered. About 2070 grains (150 grams) had been given in three days, in addition to saline enemas and hypodermoclysis. If an acidosis of any severity were present in this case, it is evident that the amount of alkaline administered was insufficient to counteract it. The alkalizing of the urine, however, indicated that sufficient alkali had been administered to overcome such acidosis as was present. The importance of this, as a diagnostic point, will be referred to again.

The blood pressure was as follows: Record of maximum and minimum blood pressure for eight months:

February and March,	140 to 220 mm.	
April,	130 to 210 mm.	
May,	120 to 150 mm.	
June,	(Period of Coma, Chart III.).	
July,	90 to 150 mm.	
August,	150 to 220 mm.	
September,	150 to 210 mm.	
October,	160 to 240 mm.	Period of hemiplegia,
November,	200 mm. Constant, Chart IV.	prior to death.

CHART I.—COMA IN COURSE OF DIABETES. RECORD PRIOR TO COMA.

Date.	Amount in c.c.	Specific gravity.	Albumin.	Casts.	Reaction.	Sugar.		Acetone.	Diacetic acid.	Urea in grams.	Symptons.	Weight in lbs.	Diet.
						Fermentation. Per cent.	Amount in grams.						
Feb. 22	2800	1038	+	+	Acid.	2.5	70	++	o	16.8	Those common to diabetes of about three years' duration. In addition, patient is feeble mentally and has arteriosclerosis. Feb. 13 and Mar. 31, slight incontinence of urine.	—	Restricted. Consisting largely of beef-juice, eggs, and milk.
" 25	1860+	1030	+	+	"	2.8	—	++	o	15.7		—	
" 27	2400	1037	+	+	"	3.5	84	+++	o	14.7		—	
Mar. 5	2400	1037	+	+	"	3.5	74	++	o	19.2		101-5	
" 7	2203	1030	+	+	"	3.7	82	++	o	22.3		—	
" 10	2450	1027	+	+	"	2.3	56	+	o	21.7		—	
" 13	2650	1032	+	+	"	2.9	77	+	o	15.2		—	
" 19	1460+	1028	+	+	"	2.6	—	+	o	19.6		102	
" 25	2550	1028	+	+	"	—	—	++	o	18.4		103-5	
Apr. 12	960+	1030	+	+	Neut.	—	—	—	—	—		—	
" 28	2480	1025	+	+	"	—	—	—	—	22.32		—	

Chart II.—Coma in Course of Diabetes. Period of Coma.

Date.	Amount in c.c.	Specific gravity.	Albumin.	Casts.	Acidity in terms 1/10 Nor. NaHO.	Reaction.	Sugar. Fermentation. Per cent.	Sugar. Polariscopic. Per cent.	Sugar. Amount in grams.	Acetone. Per cent.	Diacetic acid. Per cent.	β-oxybutyric acid.	Indican.	Total grams N. in 24 hours.	Ammon. per cent. of total N.	Total amt. ammon. N in grams in 24 hrs.	Symptoms.	Treatment.	Diet. Total fluids.
June 3	1500	1030	+	+	–	Acid	–	–	+	–	–	–	–	–	–	–	Onset of coma; irritability; weakness; muscular pains; asleep in chair most of day; arouses and converses as persons approach; refuses to save urine or obey orders; diarrhœa.	Plan for alkaline treatment; sod. bic., gr. xxx every hour by mouth; saline enemas, Oj every 6 hours; hypodermoclysis, saline 500 c.c. b. d., till urine is alkaline; intrav., and subcut. sod. bic. if condition is worse.	Milk and water 38 ounces.
" 4	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	Developing coma; asleep most of the day; responds only when shaken, then lapses into former state; no Kussmaul respiration.		Milk and water 54 ounces.
" 5	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	Coma established; cannot be aroused; incontinence of urine, feces; no Kussmaul respiration at any time.	Alkaline treatment begun; sod. bic., gr. xxx every hour by mouth, 23 doses (690 gr.); saline enemas, Oj every 6 hours (2 qts.); hypodermoclysis, saline (500 c.c.).	Milk, 34 oz. Water, 110 oz.
" 6	1680	1030	+	–	–	Acid	–	–	+	+	+	–	–	–	–	–	Coma continues; somewhat less marked; incontinence urine; specimen obtained by catheter.	Sod. bic., gr. xxx every hour by mouth, 23 doses (690 gr.); saline enemas, Oj every 6 hours (2 qts.).	Milk, 38 oz. Water, 36 oz.
" 7	1478	1028	+	+	–	Acid	–	1	14.78	0.00193	0.00048	0	0	–	–	–	Coma lessening; heavy but can be aroused; "sleeps" all day.	2070 gr. sod. bic. by mouth; 6 qts. saline enemas; 500 c.c. saline subcutaneously in 3 days.	Milk, 30 oz. Water, 42 oz.

+ = present; o = absent; — = examination not made.

Date.	Amount in c.c.	Specific gravity.	Albumen.	Casts.	Acidity in terms. 1/10 Nor. NaHO.	Reaction.	Sugar. Fermentation. Per Cent.	Sugar. Polariscopic. Per Cent.	Sugar. Amount in grams.	Acetone. Per Cent.	Diacetic Acid. Per Cent.	β oxybutyric acid.	Indican.	Total grams N. in 24 hours.	Ammon. per cent. of total N.	Total amt. ammon. N. in grams in 24 hrs.	Symptoms.	Treatment.	Diet. Total fluids.
June 8	1680	1022	+	+	—	Alk.	2.5	—	45.36	+	+	o	—	—	—	—	Coma clearing: sleeping lightly all day.	Sod. bic., gr. xxx by mouth, 3 doses (90 gr.); saline enemas, Oj every 6 hours (2 qts.).	Milk, 52 oz. Water, 40 oz.
" 9	1350	1009	+	+	2.5 c.c.	Alk. lit. acid phen.	0.69	1	13.50	0.0116	0.2299	o	o	6.04	10.6	.641	Coma clearing: brighter but still drowsy.	"	Milk, 18 oz. Water, 66 oz.
" 10	1410	1009	+	+	0.6 c.c.	Alk. lit. acid phen.	0.46	0.52	7.32	0.00348	o	o	o	5.66	8.3	.470	Coma clearing: brighter falls asleep when left alone.	"	Milk, 62 oz. Water, 12 oz.
" 11	1920	1010	++	+	3.0 c.c.	Alk. lit. acid phen.	o	0.68	13.05	0.00464	o	o	o	6.23	13.8	.860	Coma clearing: can be aroused easily.	"	Milk, 72 oz. Water, 2 oz.
" 12	1710	1014	+	+	2.8 c.c.	Alk. lit. acid phen.	0.69	0.66	11.7	o	o	o	o	8.85	14.0	1.22	Disappearance of coma: tried to get out of bed.	"	Milk, 48 oz. Water, 20 oz.
" 13	1860	1009	+	+	1.6 c.c.	Alk. lit. acid phen.	—	0.24	4.46	o	o	o	o	6.67	18.5	1.21	Absence of coma: bright; got out of bed.	Sod. bic., gr. xxx by mouth, 3 doses (90 gr.).	Milk, 61 oz. Water, 14 oz. 2 eggs.
" 14	1833	1015	+	+	1.8 c.c.	Alk. lit. acid phen.	1.8	1.86	32.9	o	o	o	o	16.9	14.5	2.3	Absence of coma: quite rational.	"	Milk, 86 oz. Water, 46 oz. 2 eggs.

+ = present; o = absent; — = examination not made.

Report Subsequent to the Coma.—From June 15 to October 24, 1907, the patient showed no evidence of coma. She remained well and active, assisting about the ward; her mental condition was such as had been noted on admission. On October 24, 1907, she again lapsed into a drowsy condition. Acetone, which had been present all along, disappeared from the urine; diacetic acid did not appear. The amount of sugar remained unchanged. Albumin and casts continued to appear as on previous examinations, with some increase in the albumin. Albuminuric retinitis was found. The blood pressure at this time will be seen in Chart IV.

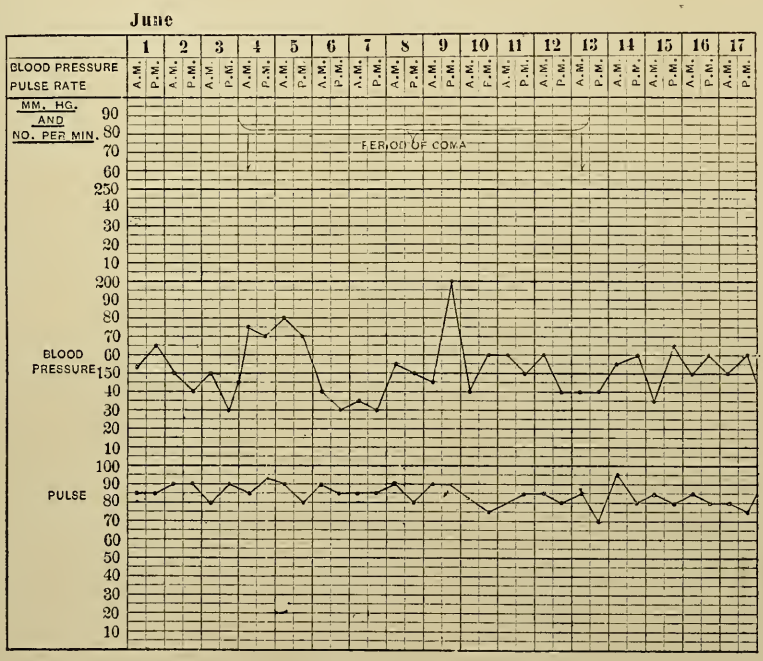

June
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17
BLOOD PRESSURE
PULSE RATE
A.M. P.M.
MM. HG. AND NO. PER MIN.
PERIOD OF COMA
BLOOD PRESSURE
PULSE

CHART III.—Blood pressure chart during June, period of coma. Compare this chart with that for October and November (Chart IV.).

On October 25, 1907, definite hemiplegia developed and continued until death occurred, November 20, 1907.

Autopsy by Dr. Horst Oertel (Report in Brief).—Female body, about forty-eight years of age, 170 cm. long; long, slender frame, fair amount of emaciation; skin, pale, clear; pupils: left, oval, medium size; right, dilated, round; there is a small arcus senilis.

On opening the thorax the left pleural cavity is dry and free from adhesions; the right pleural cavity is dry and contains a few adhesions over the apex and over the lower lobe in the axilla.

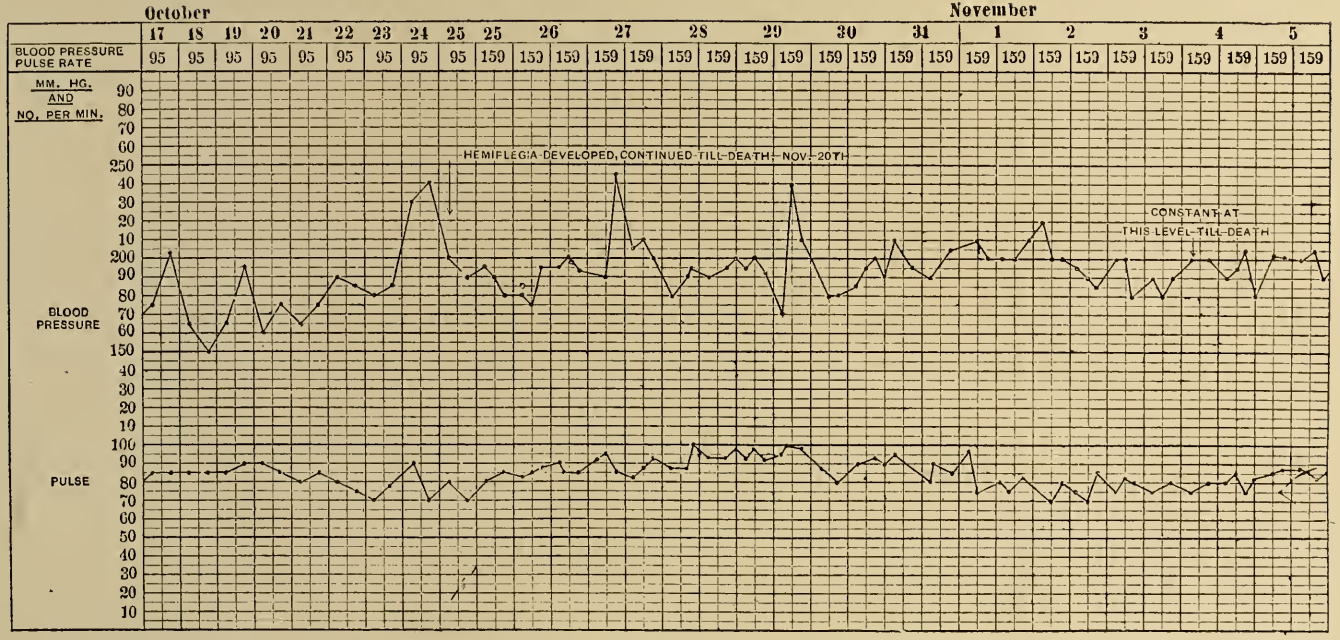

October
November
17 18 19 20 21 22 23 24 25 25 26 27 28 29 30 31 1 2 3 4 5
BLOOD PRESSURE PULSE RATE
MM. HG. AND NO. PER MIN.
BLOOD PRESSURE
PULSE
HEMIPLEGIA DEVELOPED, CONTINUED TILL DEATH, NOV. 20TH
CONSTANT AT THIS LEVEL TILL DEATH

CHART IV.—Blood pressure for October and November; period of hemiplegia, just prior to death. Compare blood pressure chart of this date with that of June, period of coma (Chart III.).

Heart.—Mitral valves: Posterior valve cusp shrunken; anterior contains large, irregular patches, which are yellowish and have wavy raised edges. The aortic valves are wrinkled and slightly stiffened at their bases. The aortic valves above contain numerous gamboge-yellow raised patches. The coronaries are small, tortuous, and contain numerous deep, yellow, raised patches. The heart muscle on section is brownish with yellowish mottled areas; the muscle bundles are not distinct; in place there are small white areas.

Left Lung.—The lung is small, its anterior portions crepitant, its posterior portions bluish, wet, œdematous. There are a few subpleural calcified tubercles, which are surrounded by black anthracotic areas. Upper lobe is crepitant, dry. The pleura at apex is negative. The lower lobe is firmer, wet, and exudes a small amount of frothy serum. The smaller bronchi are slightly reddened.

Right Lung.—The right lung is larger; the upper lobe contains a few puckered pleural thickenings, and on section it is reddish and exudes on pressure frothy serum. Some of the smaller bronchi exude purulent material, and the lobe contains scattered areas which are firm, but from which cut section sinks. The middle lobe is crepitant, emphysematous. The lower lobe is dark red, and contains scattered areas of consolidation, and exudes on pressure frothy serum. The smaller bronchi, especially those of the lower lobe, are bathed in mucopurulent material.

Spleen.—Negative.

Left Kidney.—Weight, 185 grams. The kidney is quite large, surrounded by a small amount of perinephric fat; it measures 13 x 7 x 4 cm. The capsule strips with some difficulty, leaving a finely, and in places roughly, granular surface. The lobulations are fairly well marked, and in places contain whitish puckered scars. On section the cortex has an average thickness of 10 mm.; it is yellowish and greasy; the Malpighian bodies not at all distinct. The medulla is reddish in color and the line of demarkation well preserved.

Right Kidney.—Weight, 180 grams. Same as left.

Duodenum.—Small, with well-marked rugæ. The papilla is prominent and exudes fluid bile on pressure. Common, cystic, and hepatic ducts free.

Liver.—Weight, 1500 grams. Its surface is smooth, and contains mottled yellowish areas. On section it has a somewhat greasy surface. The centres of the lobules appear dark red in color. The periphery of the lobules is light yellow.

Pancreas.—On removing the pancreas, which is rather small, the tail of the organ not reaching the hilum of the spleen, the weight is found to be 100 grams. The organ measures 17 cm. from head to tail. Its middle part is 5 cm. broad and 3 cm. thick. It is surrounded by very little fat.

On cut section, firm; the lobules are irregular in size, pinkish, and separated widely by bands which have a butter-like consistency. The pancreatic duct is apparently surrounded by a large amount of connective tissue. The splenic artery shows moderate atheroma. The pancreatic, common, and hepatic ducts and the gall-bladder are free from stones. Microscopic section shows interstitial and marked interacinar pancreatitis; sclerosis of and diminution in the number of islands; great destruction of pancreatic lobules, bringing islands into clumps.

Brain.—The brain is small, there being apparently atrophy of both frontal lobes. The vessels at the base of the brain are small, tortuous, and filled with atheromatous plaques. On opening these vessels the lumen gaps and the walls are clotted. On section of the brain a small area of softening is found in the right internal capsule along the motor tract. This area is elongated, being about 5 mm. by 1 cm. It is entirely limited to the motor tract. There is also considerable fluid in both lateral ventricles. The brain substance around this area is very soft. In the right pons is also a small area of softening about 4 mm. in diameter, well localized. Along the motor tracts the spinal cord grossly shows no area of degeneration. The posterior spinal vessels, however, are thickened and tortuous.

Etiology of the Coma.—1. Acidosis or Acid Intoxication. At the annual meeting of the Association of American Physicians held in 1907, Dr. Folin[2] presented a review of the work done on acid intoxication, and Dr. Joslin,[3] at the same meeting, presented a similar paper on acidosis. From these two papers it appears that, though for the last fifty years much work has been done in Germany and in this country on the high ammonia content and organic acids in diabetes, there is as yet no clear knowledge of either the source of these acids or the part they play in the graver stages of this disease. The chemical features, however, of these conditions are now well established, and from the record here given it can be stated definitely that neither existed in this case. The ammonia remained unusually low throughout the coma, and the diacetic acid was present in small quantity only; β-oxybutyric acid did not appear at any time (Charts I and II). That it would be incorrect to assume the absence of acidosis upon this latter negative finding alone has been pointed out by several authors. Dr Lusk, with Dr. Mendel, reported a case in which there was absolute intolerance for carbohydrates, and yet for three weeks no β-oxybutyric acid could be detected in the urine, and there was a maximum output of but 0.8 gram. of acetone daily. But this, together with the low ammonia, small total output of urine in twenty-four hours, and prompt alkalizing of the urine with

[2] Trans. Assoc. Amer. Phys., 1907, xxii. 256.

[3] *Ibid.*, p. 246.

small amounts of alkali (see discussion of treatment below), indicates the absence of acidosis or acid intoxication in this case.

2. Uremia.—The urine showed nothing in regard to nephritis, which does not occur in many cases of diabetes. Albumin was present throughout. The great increase in the characteristic casts described by Kulz [4] did not occur at the time of the coma, though casts were at all times present. The most significant finding was the blood pressure (Charts III and IV). The blood pressure ran moderately low during the coma, but in the final attack, which was associated with hemiplegia, the pressure rose and remained uniformly high until death. Albuminuric retinitis showed definitely that nephritis was present. Autopsy showed a chronic diffuse parenchymatous nephritis, which, although indicated by the urine and retinitis, could not, from any clinical manifestation, be held accountable for the coma. Beyond the blood pressure changes and the absence of increase in albumin, we must consider that there were no clinical signs by which uremia as a cause of the coma could be included or excluded.

3. Brain Softening.—This patient showed from the beginning mental weakness. There was a history of syphilis and evidences of general arteriosclerosis. This evidence was in hand at the time of the onset of the coma. Cases in literature are not lacking [5] to show the frequent association between diabetes and encephalomalacia. Four weeks before death hemiplegia developed, and definite evidence of this condition was found postmortem. At the time of coma, however, we had not these facts, and the stupor was different in no way clinically from graver forms in diabetes. The mental weakness, the incontinence of urine and feces, together with mild diabetes and absence of signs of acid intoxication, were important facts pointing to the central nervous system. The subsequent hemiplegia was very significant. There is no explanation as to how coma, in such brain conditions, is produced.

4. Disturbed Proteid Metabolism.—Although nitrogen partition was made in this case, it seems inadvisable to attempt conclusions from one case. This portion of the record, therefore, is omitted.

Alkaline Treatment.—This, while of no value therapeutically in the present case, proved to be so diagnostically. It will be remembered that treatment, as recommended by Stadelman and followed by Naunyn and Magnus Levy, required 100 grams of bicarbonate of sodium a day. As high as 80 grams have been injected intravenously, the reaction of the urine being the guide to the quantity to be administered. In the case here reported 150 grams by the mouth in three days was sufficient to alkalize the

[4] Domansky and Reimann, Ztschr. f. Heilk., 1901, and Herrick, Amer. Jour. Med. Sci., 1900, vol. cxx.

[5] Frerich, Ogle, Dreyfus, etc., cited by Naunyn.

urine. This was in itself evidence of either the absence of acid formation or, if present, in quantity insufficient to account for such grave clinical manifestations as appeared.

Conclusions.—From the foregoing evidences the case seems to have been one of syphilitic arteriosclerosis (history of syphilis, presence of arterial thickening, arcus senilus, and aortic cardiac sclerosis), with involvement of the pancreas, kidney, and brain. The sclerotic changes in the pancreas (interacinar pancreatitis) led to the diabetes, those in the kidney (chronic parenchymatous nephritis) to the chronic nephritis (albuminuria, casts, and albuminuric retinitis), and those in the brain to the softening with hemiplegia, to the feeble mentality, and possibly to the coma.

At the bedside, when the coma appeared, the syphilitic history, the arteriosclerosis, the enfeebled mentality, the mild grade of diabetes, the absence of any increase in the diacetic acid, the non-appearance of β-oxybutyric acid, the low ammonia, the small quantity of alkali required to alkalize the urine, the absence of any increase in total quantity of urine in twenty-four hours, seemed to be clinical features differentiating this coma from the graver forms. The subsequent hemiplegia was very significant. The low blood pressure and absence of any increase in albumin and casts were suggestive, but not positive signs by which uremia could be excluded. The urinary findings and the small amount of alkali required to alkalize the urine seem in themselves important guides in diagnosis and safeguards against false conclusions with regard to the part played by the alkaline treatment.

The only coma in diabetes clinically defined is the dyspnœic type, and the only coma chemically defined is that due to acidosis and acid intoxication. The diagnosis of these conditions, as a rule, offers no great difficulty. It is the atypical group or that due to complications which occurs more frequently and which presents the real diagnostic and prognostic problem to the clinician. The nephritis and brain condition are most important in the consideration of the coma, but in the absence of any clear knowledge of how coma is produced in uremia or brain softening, the mere statement of their presence is not sufficient to exclude other factors resulting from disturbed metabolism (proteid for example) which subsequent investigation may disclose.

The discussion of the case with nine months' clinical record, and, finally, the postmortem findings before one is very different from the problem offered the clinician at the bedside at the onset of the coma. At that time it was impossible to say that fatal coma was not developing. I believe two important findings at that time justified one in assuming that the coma was of mild type, namely, the character of the urine and the amount of alkali required to neutralize the urine. In forty-eight hours from the onset of the

coma these features manifested themselves. While no explanation of the coma appears in the record, these clinical findings may prove to be features differentiating the comparatively common non-fatal coma in diabetes (due possibly to complication or some yet unexplained metabolic disturbance) from the rapidly fatal coma in this disease.

The laboratory examinations were done in the Strecker Laboratory under Dr. Oertel and in the Cornell Clinical Laboratory under Dr. Hastings. I wish to thank Drs. Woodruff and Groeschel, house physicians, for their interest and care in preparing the records.

Dr. Joslin kindly reviewed the records with regard to the question of acidosis, or acid intoxication, and his opinion is expressed in the following letter:

"In my opinion the case * * * illustrates coma simulating the coma of acid intoxication, and yet certainly has absolutely no connection with that form. It is essential for physicians to remember this possibility, and not to treat all cases of coma in diabetes as if due to acid intoxication. The case is further valuable because so often in the past cases of coma in diabetes have been said to occur without proving the acidosis. This case was carefully studied, acidosis eliminated, and coma proved to be of another type.

"The case was evidently mild diabetes, because the carbohydrate balance was pretty certainly, from the records given, positive rather than negative. For example, the greatest quantity of sugar was 84 grams, on February 27, and yet evidently considerable milk was used at that time, and the diet probably contained more than this amount of carbohydrates. Further, the case was evidently mild, because there was no diacetic acid in the urine during February, March, April, and only a very small quantity in June. Diacetic acid constitutes only about 10 per cent. of the total acidosis, and acetone constitutes only about 10 per cent. of the combined acetone and diacetic acid. Consequently we are absolutely certain that the acidosis was insignificant.

"For the above reasons it is not necessary to know more details of the urine during the coma, because the analyses before and after settle the severity of the case. There are a few additional factors which help to exclude the acidosis: First, the fact that the urine became alkaline on June 8, during the coma, while the patient was taking an insignificant quantity of alkali, *e. g.*, 2 grams hourly. Second, the total quantity of urine at no time exceeded 2,000 c.c. No case of diabetic acidosis, to my knowledge, has been reported without nature making an effort, by the passage of large quantities of liquid, to wash out acids from the body. Third, the increase in the percentage of ammonia was very slight, and the total quantity far too low to enable one to claim acidosis."

II.—Surgical Division

REPORT OF A CASE OF IDIOPATHIC SEPTIC PLEURISY AND SEPTIC PERITONITIS.

By Howard D. Collins, M. D.

Attending Surgeon.

Reports of cases of so-called idiopathic peritonitis have been received by some members of the profession with a good deal of scepticism, and in certain cases not improperly. On the other hand, enough cases have been reported where, in spite of a very careful autopsy, the only conclusion that could be reached was that the patient suffered from a general septicaemia of which the paramount and perhaps only evidence was an acute vicious purulent peritonitis. In other words, the blood was impregnated with streptococci that seem to pick out as their field of manifest activity, a serous membrane, preferably the peritoneum.

The following history gives the story of a patient brought under my care at the City Hospital.

Mary T., an unmarried girl 16 years of age, a native of Poland, a maid by occupation, was admitted to Dr. Camac's service on October 23, 1907. The chief complaint was swollen feet and ankles and persistent headache.

The family and previous history were negative, save that in June, 1907, she was ill for a week, during which illness her hair was cut short on account of her headaches. The history of the illness in June is indefinite and unsatisfactory, but the inference was made, on the strength of the statement of friends, that the illness was scarlet fever.

After the improvement of the June illness, the girl noticed she passed but little urine and that her feet and ankles were swollen, that she was puffy under the eyes, easily fatigued and had severe headache.

On admission to the hospital October 23, 1907, the diagnosis of post scarletinal nephritis was made.

Examination showed a well nourished girl with moderate general oedema, marked under eyes and in feet and ankles. Heart not enlarged, action forcible, slightly irregular with systolic murmurs over apex and pulmonic area.

Lungs and abdomen negative.

Urine for 24 hours 28 ounces as compared to 52 ounces fluid ingested. Qualitative tests showed heavy mass of albumin, red blood cells, no sugar. Pulse and temperature not elevated.

During November the urine ranged from 20 to 40 ounces per diem, with a general average of about 28 ounces. Temperature normal, pulse about 80.

On November 23 she complained of pain in left ear.

Examination elicited slight tenderness over the left mastoid, no redness or oedema. Left drum was white and thickened, with a perforation at lower posterior quadrant; there was a very slight purulent discharge. Temperature rose to 101 deg. F. for a few hours and then returned to normal. Pulse correspondingly increased. Beyond the slight tenderness and discharge, the ear gave no further trouble until December 19, 1907. The patient was up in a chair every day; the urine was somewhat increased in amount, but of the same general character qualitatively. On that day she suddenly had a rise in temperature to 102 deg. F.; complained of sore throat, pain in limbs, severe pain in right side of thorax and upper part of abdomen. The chest signs were normal, nothing could be made out in abdomen except general tenderness. Throat congested and tonsils somewhat swollen. During the night the temperature rose to 105.3 deg. F., pulse 134, respiration 28. The blood count showed 23,000 leukocytes 90 per cent. polyneuclear.

During December 20 and 21, her condition was about the same, with fluctuating high temperatures and leukocytosis ranging between 21,000 and 31,000; polyneuclear 89 per cent. to 93 per cent. Abdomen generally tender, somewhat less rigid. Dullness over bases of both lungs, with a few fine crepitant rales. Enemata effectual.

December 22 showed an increase in respiration to 50, pulse 136, temperature dropped to 99 deg. F. Abdomen more tender, less rigid.

At noon on December 22 I saw the patient and advised operation as I felt convinced of a general peritonitis existing. About 3 p. m. under ether, I opened the abdomen by a median incision. The entire cavity was flooded with a creamy pus, white and without odor. The peritoneum was only slightly congested and in no place had lost its lustre. A very careful but rapid examination could detect no organ as the sight of origin for the peritonitis. The abdomen was washed out and the abdominal wound loosely closed with several drains leading down to the peritoneum. Intravenous infusion was given at the time of operation. Cultures of the pus and also of the blood from the infusion wound were made and subsequently reported as containing a high count of streptococci.

The patient never rallied and died twelve hours after operation. Autopsy was done 12 hours after death. The kidneys showed parenchymatous nephritis. Heart was the seat of fatty degeneration and endocarditis. Lungs congested, no pneumonia. The left pleural cavity presented the most interesting point in the autopsy, for the cavity contained about 200 c. c. of pus similar to that of the abdomen, with no attempt at sacculation. The abdomen revealed nothing beyond what had been observed at operation. A very careful search was made to detect any macroscopic communication between pleura and peritoneum through the diaphragm or a perforation of any

of the viscera, with negative result. The left mastoid was removed in order to make a more careful examination and showed an apparently old mastoiditis confined entirely to the cells and middle ear. At no point had the bony wall been penetrated.

The cause of death was given as streptococcus septicaemia with a streptococcus pleurisy and peritonitis.

This case seems to me worthy of report, for it gave us an opportunity of watching for several weeks in a scientific institution, a patient suffering from one disease (nephritis), and observing the onset of another and following it to a conclusion.

Idiopathic peritonitis of course presupposes an entrance of the septic organism into the system at some point or other, but that the point of entrance cannot be determined.

It might be fairly claimed that in this case the entrance of streptococci was through the old mastoid disease, but it might fairly be objected to on the ground that the mastoid was well walled off, and the slight lighting up of the old trouble was of a very transient character and antedated the terminal illness by nearly four weeks.

The tonsillitis of December 19 might also be offered as the source of infection.

Still it seems to me as though this case offers as vague a point of entrance for septic bacteria as any of the cases of idiopathic peritonitis and I think is as fairly entitled to that diagnosis as most of these cases, with the additional point of interest in that the septic agent selected another serous membrane—the pleura—as well as the more commonly invaded peritoneum.

CARCINOMA OF THE SIGMOID.

By Edward Milton Foote, A. M., M. D.,

Attending Surgeon.

It was once the fortune of the writer to assist at an operation upon a man in the sixties, suffering from intestinal obstruction. He walked from his bed into an adjoining room, raised himself upon the operating table by stepping on a stool, and lay down and took the ether. When his abdomen was opened, there was a quantity of fluid fecal matter free in the peritoneal cavity. The distended and gangrenous intestine immediately above a carcinoma of the sigmoid had given way, either as the result of the patient's recent bodily effort, or a short time before. But even had this accident not occurred, it is doubtful if the patient could have survived, as the distention and paralysis of the large intestine were so extreme. Such fatal delay can

only be avoided by an earlier diagnosis—but is an early diagnosis of carcinoma of the sigmoid ever possible?

Carcinoma of the sigmoid forms less than two per cent. of all cancer.[1] Its relative importance in abdominal diagnosis is, however, somewhat greater than these figures would indicate, as it holds third place in order of frequency of gastro-intestinal cancer, the stomach coming first and the rectum second. It has an especial interest for the surgeon, since it grows slowly, and does not form distant metastases for a long time. It is also accessible, and is situated in a part which can be sacrified almost with impunity. But to enter this attractive surgical field under favorable circumstances, an early diagnosis is necessary, and the difficulties which surround an early diagnosis of carcinoma of the sigmoid are a challenge to physician and surgeon alike.

The anatomy of this region is simple. The sigmoid forms a loop about 18 inches long, with a short meso-sigmoid above and below and a longer one in the middle of the sigmoid. Its arteries are branches of the inferior mesenteric; its lymphatics drain into the lumbar glands. Its own lymphatic glands are less numerous than those of other parts of the intestine. It follows from these facts that a removal of a part of the sigmoid, and the corresponding part of its meson, is not a difficult operation; but that such an operation will be a complete removal of the carcinoma only in the early stages of the disease. When the carcinoma has spread to the lumbar glands, its complete removal will be impossible.

Three Personal Observations.

Before taking up the symptoms upon which we must rely to make an early diagnosis of carcinoma of the sigmoid, let me give very briefly the histories of three patients upon whom I have operated at different times for this disease. The first was a man aged 53 who, for six months had suffered from increasing constipation, with a good deal of pain in the lower left abdominal quadrant. The pain and constipation had at times been much worse than at others, so that on several occasions the patient had been threatened with complete obstruction, if one may so express it. He had not observed any blood or mucus in the stools. There had been a gradual, but progressive, loss of flesh and strength, but up to the occurrence of an acute obstruction for which I was consulted, he was able to attend to business every day. When I first saw him there had been no movement of the bowels for four or five days, and there was gradually increasing vomiting, abdominal distention and pain. He was still in good general condition. In other words, he had the symptoms of a complete obstruction of the large intestine low down, for the relief of which immediate operation was indicated.

1. Heimann in v. Bergmann's System of Surgery.

I held the hope and expressed it to the family that the cause of obstruction might be purely mechanical, such as a volvulus. In the light of subsequent experience I knew such a hope to be absolutely groundless, for the man's history, though less exact in details than we should like it to be, is typical of carcinoma of the sigmoid. Operation revealed that the obstruction was due to such a growth; that the disease had involved the whole wall of the intestine, and had also attached to it two loops of small intestine. An artificial anus enabled the patient to go about in comparative comfort for some months, and postponed his death for more than a year.

The second patient was a woman nearly fifty years of age, who was slowly dying from recurrent carcinoma of the breast. She had the same abdominal symptoms as the man just spoken of. There was gradually increasing constipation, with occasional acute attacks, going on almost to complete obstruction. As a tumor was palpable there was no difficulty in establishing the diagnosis. She would not consent to operation, however, until complete obstruction occurred. By that time the growth in the sigmoid had already become attached to the bladder, so that complete removal was out of the question, even if she had not had a recurrent carcinoma of the breast. An artificial anus was established which performed its functions well until death, about six months later.

These palliative operations are a physical relief to the patient, but they bring little credit to the surgeon, since the attention of the patient and his family is fixed, not on the pain which he has escaped, but on the sad fate which is before him.

The third patient presented a more pleasant surgical task.

Mrs. L., 55, a stout Irish widow, not unfamiliar with beer and whiskey, had always been healthy. For some months previous to her admission to the New York City Hospital she had suffered with constipation, rarely having more than two movements a week, and often only one. The stools were small and hard; otherwise normal as far as she had noticed. Associated with the constipation was a feeling of itching or burning in the lower left side of the abdomen. One night she awoke with cramps in the abdomen. There had been no movement for two days. This was not unusual, but she did not like the severe cramps, and she took, the following day, in order to relieve herself, citrate of magnesia, seidlitz powders and salts. They produced vomiting, but no movement of the bowels. The vomiting was a new symptom. In one of the medical wards of the hospital, enemata and additional cathartics failed to effect a passage, and the question of surgical interference was raised.

When I saw the patient she was still in good condition, with no fever and a good pulse. It was then five days since the last movement of the bowels. The abdomen was uniformly distended to a moderate degree and

tympanitic. There was no localized pain nor tenderness nor muscular rigidity, and no tumor was palpable. It was evident that the patient was suffering from complete obstruction, very low down in the large intestine. Rectal examination revealed nothing abnormal, but the fact that the patient could hold only a pint of fluid given by rectum, confirmed the theory that the obstruction was low down. Peristaltic motion was still active as shown by the rhythmic cramps, as well as by auscultation, but no gas passed the anus. The diagnosis made was stricture of the sigmoid, probably malignant, and operation was performed at once.

An incision was made at the outer border of the left rectus. The moderately distended small intestines filled the field of view. The upper part of the sigmoid was brought into the wound, and was not greatly distended. By following it downward, the finger came upon a hard mass, of scarcely larger caliber than the normal intestine, and not adherent to any surrounding part. It was so low down, however, that it could only be brought out of the wound by dividing a part of its mesocolon. It was a constricting tumor of the sigmoid, and it had already penetrated the serous coat, where it was growing in little red masses characteristic of a carcinoma.

The affected intestine and a considerable portion of healthy intestine, both above and below the tumor, was freed by division of its meson, and brought well out of the wound. The peritoneal cavity was closed around it, and the loop of intestine wrapped in gauze. Owing to the necessary division of blood-vessels, nearly the whole of this displaced intestine became gangrenous. This had no effect on the subsequent steps of the operation, but spoiled the pathological specimen.

A week later the surplus intestine was excised and the ends sutured. Owing to the rigidity of the intestinal wall, it was difficult to properly approximate the two ends, and the suture did not hold. Another plan was then adopted to re-establish the continuity of the intestine. The intervening septum, or spur, was cut through by crushing it between the jaws of a large curved clamp. This was done three times at intervals of a few days, until a deep division of the septum was obtained. Only at the third application of the clamp did the patient feel any pain. The opening in the intestine was then closed once more by suture, and this time the stitches held. A small fistula still remained, which healed spontaneously, as there was an ample lumen at the point of resection and suture.

This course of operative treatment occupied about three months, or nearly six months, if one reckons the time until the fistula was finally healed.

Analysis of Early Symptoms.

Carcinoma of the sigmoid occurs in both men and women. The age of the patient is usually between forty and sixty years.

Early symptoms naturally fall into three groups. There are the local symptoms of pain, tumor, and abdominal distention; the general symptoms of altered stools, less of flesh and strength, and cachexia; and a third group of symptoms due to the presence of partial or complete obstruction of the bowel.

Of the local symptoms, pain is the most important and usually the first noted. It is referred to the lower left quadrant of the abdomen. It varies greatly in amount in different cases. It may be slight or severe. If present at all, it is more or less steady until the tumor begins to interfere with the passage of the feces through the sigmoid. Then the pain comes on in rather distinct waves as the colon, by increased peristalsis, tries to force its contents through the narrowed sigmoid. Both kinds of pain, the steady kind and the peristaltic kind, are much more marked at times when the fecal masses are hard and accumulate in unusual amount.

The presence of a tumor is not usually observed at an early stage of the disease. Indeed, when it occurs early it is rather suggestive of non-malignant disease as is mentioned in a later paragraph on differential diagnosis.

Abdominal distention in an early stage of the disease is associated with partial obstruction of the bowel. Otherwise it is not of common occurrence, and has little diagnostic value, being found in too many other conditions.

Increasing constipation is an almost constant symptom. Taken with the pain it forms the most reliable basis for early diagnosis. But the constipation does not increase steadily. On the contrary, there is usually an irregularity in the increase so that one may well speak of acute attacks of constipation, superimposed upon a chronic constipation. That is to say, the movements of the bowels will become scanty and infrequent, and perhaps cease for three or four days. Then as the result of cathartics or enemata, there will be three or four movements, the last probably loose. After this the bowels will act satisfactorily for a few days or possibly for some weeks. Then another attack of constipation comes on. Inquiry will show that these attacks of constipation tend to occur at shorter intervals and that stronger cathartics are required to overcome them.

Alternating constipation and diarrhea is a natural development of this increasing obstruction, but it is usually not well marked until an ulcer forms at the site of the tumor, or in the damaged intestinal wall above it. Such spontaneous diarrhea following a period of constipation is not to be confused with the loose movements following the administration of strong cathartics to overcome the constipation. Such movements cannot properly be classed as a symptom of the disease, and care should be taken to exclude them before making the statement that alternating constipation and diarrhea exist.

The characters of the stools themselves are seldom of assistance in making an early diagnosis. Blood and mucus are rarely seen at an early stage.

It is important to remember this, as blood in the stools is usually mentioned as one of the symptoms of carcinoma of the sigmoid. It is a symptom of the ulceration and therefore a later symptom. We are considering here the early diagnosis. One should never decide that a patient has not a carcinoma of the sigmoid because the stools do not contain blood. Many patients who have such cancer go on to complete obstruction or even death without visible blood in the stools. The occurrence of blod and mucus in the stools is closely associated in time and origin with the alternating diarrhea and constipation.

Loss of weight is an early symptom, though like cachexia, it is more noticeable at a later stage of the disease. Those who make a practice of weighing regularly all of their patients, know that loss of weight is an early symptom of many obscure diseases.

It seems rather odd to class obstruction as an early symptom, but we are justified in doing so, both pathologically and clinically. Carcinoma of the sigmoid is usually annular and often it gives symptoms of partial or even complete obstruction before it has involved more than an inch or two of the bowel. This was so in the third of the cases mentioned at the beginning of this paper. Clinically it must stand at present as an early symptom, for many of these patients do not consult us until they suffer from some form of obstruction. Yet many of them do come earlier for pain or constipation or malaise, and we fail to realize the possibilities that underlie these symptoms.

If obstruction occurs, it is of the mildest type possible. For a time some gas may pass the constriction and serve to postpone the marked abdominal distension which is later a feature of all complete obstruction of the large intestine. If the stricture is situated as low down as the sigmoid, complete obstruction may exist for several days before there is vomiting or serious pain or distress. This is, of course, due to the fact that the patient has nearly the whole of his large intestine as a temporary reservoir for gas and feces, so that if he eats little it may be some days before the small intestine and stomach show signs of the obstructed flow.

Physical Examination.

The conditions found upon examination are quite different when an acute obstruction is present and when it is absent. If the patient is seen between the attacks of acute constipation, when the large intestine is not overfilled, examination of the abdomen will reveal tenderness in the left iliac fossa, especially when rectal and abdominal examination are combined. If the patient is thin and a growth is situated in that portion of the sigmoid which is normally palpable, a tumor should be felt at a very early stage of its development. If the patient is stout, or if the growth is situated in the

lower part of the sigmoid, a tumor cannot be felt at an early stage. The tumor, if felt, will be small and movable at an early stage of the disease. When the growth has reached the serous coat of the bowel, adhesions form which limit its mobility. A large or immovable tumor palpable within a few weeks after the first symptoms are noticed by the patient is probably non-malignant; a point which will be again referred to in connection with differential diagnosis.

The rectum is to be examined with the finger and through a tube. If a tumor can be felt, it is of the rectum and not of the sigmoid. Inspection through the endoscope may reveal merely a constriction, or a tumor, or an ulceration secondary to the cancer, or nothing abnormal. Failure to find even a constriction does not prove its absence, since only a little more than the lower half of the sigmoid can be seen under the most favorable circumstances. The tube used for this inspection should be not less than twelve inches long, though in many cases not more than eight or ten inches can be passed. A simple tube and obturater, with light reflected from a head mirror will suffice, if one has not at hand the more elaborate style of endoscope, with pneumatic and electric attachments.

Another method of examination which should not be omitted is the pumping of air or water into the rectum and observing resulting changes in percussion and auscultation. Sometimes a distinct gurgling marks the site of constriction.

If the patient is seen during an attack of acute obstruction, the abdomen is distended with gas at least sufficiently to defeat palpation. There may be visible peristalsis. There may be an area of dullness in the left lower quadrant, due to the accumulation of fecal matter above the constriction. Water or air injected into the rectum will probably not pass the constriction. In this case not more than a pint of water can be injected, as this is about the capacity of the rectum. The sigmoid, it will be remembered, holds about a quart, and when the carcinoma is high up in the sigmoid, some of this capacity is available, even though obstruction is complete.

Differential Diagnosis.

Chronic Constipation—In making a differential diagnosis, we shall have to consider first of all chronic constipation. This often shows a tendency to increase as a patient passes middle life, and either on account of injudicious diet, or lack of exercise, or for some other reason, it varies in intensity, and may even be interrupted by short periods of diarrhea. It may also be accompanied by a diminished appetite, poorer color, and a loss of weight. The chief points which distinguish chronic constipation from the irregular intestinal action due to malignant disease, are the long duration of symptoms and the absence of abdominal pain, except such colic as may accompany an attack of diarrhea. In the simple non-malignant cases of constipation the

symptoms will have lasted for years. However, it is well to remember that a carcinoma of the sigmoid may develop in a person who has long been constipated from some other cause.

Sigmoiditis—The occurrence of an acute or subacute inflammation of the sigmoid was formerly denied by some of the most prominent writers. Whether they have all retracted or not it is not necessary to inquire. For our purpose it is enough to know that many cases have recently been reported, in some of which the diagnosis has been confirmed by operation. Without going into the various classifications of such inflammation which some writers have proposed, we may also admit that the various forms of catarrhal ulcerative and perforative inflammation may occur in this region. Such an inflammation may vary in intensity, and it may be confined to the mucous membrane or penetrate the deeper layers of the intestine, or extending through the intestinal wall the inflammation may lead to the development of localized abscesses, sinuses and fistulae. It is evident that the symptoms in different cases will vary a good deal, and yet it is possible to speak of the typical set of symptoms. The inflammation is usually chronic with acute exacerbations. In the acute stage there is fever with tenderness and muscular rigidity, colisky pain, and perhaps fetid or purulent stools. In the subacute or chronic stage, the patient may be constipated or there may be diarrhea with blood and mucus in the stools. There is more or less constant pain. The sigmoid is palpably thickened and tender, for a distance of several inches. Such a tumor is often movable. It does not disappear promptly after an evacuation of the bowels is obtained. The colon above the sigmoid is often distended. A marked restlessness or nervousness has been observed in some patients. Naturally in these cases there is often a considerable loss of weight, which may be quickly recovered, upon a suitable diet. If under treatment the inflammatory symptoms subside, the patient gains in weight and the stools become normal, the suspicion of malignancy is lifted. If an ulcer of the sigmoid leads to perforation, usually through one of the diverticula, and the abscess which results is limited either by adhesions, or because it forms retroperitoneally, the patient will present some symptoms similar to those of malignant disease, but the picture is so essentially inflammatory that a careful investigator is not likely to be misled.

Tuberculosis and Syphilis—Tuberculosis and syphilis of the sigmoid are so rare that a brief mention of them must suffice. The former leads to adhesive inflammation and sinuses; the latter has its own history in many cases, and generally other lesions in other parts of the body.

To sum up the chief points in differential diagnosis we may say,

If the disease is located in the left iliac fossa, with symptoms of increasing constipation for six months or less, possibly with occasional diarrhea without blood, with local pain and some impairment of strength, there is

strong suspicion of malignancy, especially in a person of middle age or beyond. If a small movable tumor can be felt the diagnosis is almost certain.

If there is a history of diarrhea and constipation, with mucus and blood almost from the beginning of the illness, or if the history includes an acute inflammatory attack; or if there is a large movable tumor, or a continuation of symptoms for a year or two without great impairment of the health, the affection is probably not malignant.

Complete Obstruction.

If the patient when seen is suffering from complete obstruction, it will be necessary to consider in addition to the possibilities mentioned above, mechanical obstruction, such as volvulus of the whole sigmoid, obstruction due to bands, etc. The absence of symptoms previous to the occurrence of the acute obstruction will usually serve to differentiate the two kinds of obstruction; but should a mistake be made in this direction, it is not serious, as the treatment indicated is in both cases immediate operation.

Treatment.

It is not the purpose of this paper to treat the subject of carcinoma of the sigmoid exhaustively, but rather to suggest the possibilities of an earlier diagnosis than is usually made. The remarks upon treatment will, therefore, be very brief.

It has for many years been recognized that primary resection of the large intestine followed by closure of the abdominal wound is a dangerous procedure, even when the intestine contains only the normal amount of fecal matter. There are several reasons for this, which are not necessary to mention in detail, but the chief one is undoubtedly the absence of peritoneum from a considerable portion of its circumference. The risk of resection is greatly increased if performed in the presence of partial or complete obstruction.

As long ago as 1879, Baum[2] proposed and carried out a plan to lessen the risk. His idea was to perform colostomy some distance above the tumor in order to relieve the distention, if such existed, and also to divert the fecal stream. Some days later he performed a resection, and the sutured intestines had time to unite before any feces passed through it. This plan of procedure has been followed by many surgeons and is still in use to-day. If the tumor is situated in the sigmoid, the artificial anus is usually established in the transverse colon.

In 1892 Bloch[3] performed what he called an extra-abdominal resection of the sigmoid, dividing the operation into three steps or stages. At the

2. Centralblatt für Chirurgie, 1879, p. 169.
3. Centralblatt für Chirurgie, 1892, p. 628.

first one the tumor was brought out of the abdominal wound, and the intestine was opened above the constriction. At the second step, taken some days later, the protruding intestine was resected. The operator then waited four months to make sure that there was no local recurrence of the tumor, and then performed the third part of the operation, which consisted in freeing and suturing the edges of the intestine.

This method of operating seems to possess certain distinct advantages over Baum's method, especially in cases of carcinoma of the sigmoid. The technique was perfected by Mikulicz,[4] and a description of it was published by him in 1902. His plan is to free the tumor, and the adjacent involved lymphatics, dividing so much of the meson as is necessary to bring the affected intestine out of the wound. The edges of the parietal peritoneum are stitched to the upper and lower portions of the intestinal loop with cat-gut in order to close the peritoneal cavity, and to prevent the intestine from drawing back into it. The wound in the skin is also partially sutured. In his earlier operations Mikulicz delayed resection for one, two or three days. In his later operations he cut off the protruding loop of intestine at once, and tied in its upper opening a large tube in order to convey the feces away from the wound. This immediate resection is undoubtedly the proper procedure to follow, for the loop often becomes gangrenous in part, owing to the disturbance of its blood supply. Two or three weeks later, Mikulicz crushed the septum or spur between the upper and lower openings in the intestine, and closed the external intestinal wound by suture. This reduces the operation to two steps, unless it is necessary to repeat the second one.

Performed in the manner last described, resection of the sigmoid flexure is almost without risk, whereas by Baum's method of operating, there is some risk of peritonitis, even when an empty intestine is resected and sutured. Baum's method also requires two abdominal wounds. The chief objection which may be urged against Bloch's extra-abdominal resection is the possibility that it may be followed by hernia. If this occurs, a third operation will be required to cure it, but as the intestine need not be opened at this operation, it does not add greatly to the risk. All things considered, it is a most satisfactory method of operating for carcinoma of the sigmoid, and deserves a wider recognition than it enjoys at present. There is one point in connection with crushing the septum which I have not seen mentioned, but which is worth observing. Before the intestine is sutured to the parietal peritoneum, its upper and lower portions should be slightly rotated upon themselves, so that their peritoneal surfaces will lie in contact. If this is done, the septum will contain no sensitive nerve fibres, and may therefore be crushed absolutely without pain.

4. Centralblatt für Chirurgie, 1902, Beilage, p. 106.

CASE OF RHABDOMYOMA SARCOMATODES OF THE TONGUE.

WITH REMARKS ON THE USE OF TRYPSIN AND AMYLOPSIN IN THE TREATMENT OF MALIGNANT DISEASE.*

FREDERICK HOLME WIGGIN, M. D.,

Consulting Surgeon.

Primary sarcoma of the tongue is a disease so seldom met or recognized and recorded that a case of this character presents features of unusual interest. There appear to be less than forty well authenticated cases on record. Of the various varieties of sarcoma invading this organ, the one under consideration seems the least frequent, no recorded case having been found in the literature available.

History—N. M., an unmarried female, aged 23, of Irish descent, a domestic, applied in November, 1905, to the Metropolitan Nose and Throat Hospital for treatment, complaining of an enlargement of the back of her tongue, which was causing her much discomfort, as, on account of its size, it made speaking, eating and sleeping difficult. The patient then stated that she had asked for treatment at several hospitals in London, but had been refused operation.

She was referred to me as one of the consultants of the hospital for an opinion as to the nature of the growth, and the method of treatment to be employed. She gave a negative family, as well as personal history, up to about seven years previously, when she stated that she had first noticed a small growth projecting from the surface, at the back of the tongue, round at the bottom, and somewhat pointed at the top. This was shown to a physician, who pulled it off and treated the resulting sore for a time, and then as the growth began to reappear, left it alone. The patient stated that it grew rapidly for a time and then more slowly, but that of late, the tumor had been enlarging rapidly; she also said that she had had no pain, other than that caused by the mechanical difficulty encountered in speaking, eating and sleeping, especially the latter, as for some time past she had been obliged to sleep in a sitting posture.

Examination—The girl appeared to be well nourished, but anemic, and rather dull mentally. Examination of the heart, lungs and abdomen failed to reveal anything of an abnormal character; when the mouth cavity was looked into, however, a tumor which nearly occluded the pharynx was found, projecting from the surface of the tongue at the site of the foramen cecum, rather more prominent to the left of the median line than elsewhere

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and about two and one-half inches by one inch in size. The growth was painless to the touch, but felt elastic almost to the point of fluctuation. The mucous membrane covering the growth was intact and normal in appearance and no glandular enlargement could be made out.

Diagnosis—Taking into consideration the sex and age of the patient, the location of the tumor, its elasticity and the condition of the mucous membrane covering it, I considered it probable that the neoplasm was made up of thyroid tissue and its removal was advocated.

Operation—In accordance with this advice, the patient entered the French Hospital and on November 24, 1905, I performed the following operation, with the assistance of Dr. James Tayloe Gwathmey: Dr. Gwathmey anesthetized the patient by his vapor method, by means of a long hollow metal tube of the size and shape of a number 15 English catheter with its end cut off, the anesthetic vapor being delivered directly into the larynx. This instrument was also employed to retract the tissues, being introduced into and along the side of the mouth nearest the field of operation. A heavy silk suture was then passed through the tongue, just beyond its tip and, by means of this retractor, the tongue, which was freely movable, was drawn well forward and a longitudinal incision, one and a half inches long, was made through the mucous membrane and muscular tissue covering the most prominent portion of the tumor, which was, as has already been stated, to the left of the median line. The tissues were easily reflected from the tumor, which was found to be encapsulated and which was readily shelled out of its bed. It was about one and a half inches in diameter and when the growth was cut into, it was found to consist of soft, fatty looking tissue. The incision in the tongue was closed by suture, which readily controlled the existing hemorrhage which was slight. No effort was made to remove the remaining enlargement on the right side, which had somewhat subsided with the removal of the tumor already mentioned, it being thought that it was due to the pressure exercised on the tissues by the tumor just removed and that in time it would disappear.

Result—The convalescence proved uneventful and the patient improved very much in general health and in appearance, being now able to sleep in the prone posture, and to swallow with comparative comfort, but her ability to speak distinctly did not improve materially.

Examination of Tumor—The tumor was submitted for microscopic examination to five eminent pathologists, with the result that three gave opinions favoring fibroma and two fibrosarcoma. Dr. W. R. Williams, pathologist of the French Hospital, through his assistant, Dr. Krumweide, reported as follows: "Specimen removed from tongue of N. M. is histologically a fibroma, the softness of the tumor is due to edema of the tissues.

Though somewhat cellular there is no sarcomatous appearance and it is therefore benign."

Prof. E. K. Dunham, of the New York University and Bellevue Hospital Medical College, said: "The specimen of lingual tumor which you sent to me is a fibroma, in most places it is composed of dense fibrous tissue, in other places the tissue is more areolar, the surface of the specimen presents fibres parallel to the surface, apparently due to compression of the surrounding structures. There are only a moderate number of vessels in the specimen and these are of moderate size. There is no suspicion of malignancy."

Dr. F. M. Jeffries informed me that Prof. James Ewing, of Cornell University, had also seen the specimen and concurred with him in his view that the tumor was a fibrosarcoma. As the opinions of the pathologists who had examined the specimen were now equally divided, it was thought to be of sufficient interest to obtain the opinion of Professor William H. Welch, of Johns Hopkins University, and a specimen of the tumor was accordingly sent to him; the following was his reply: "I do not generally quarrel over differences in diagnosis between fibroma and sarcoma, as it is a question ordinarily of richness in cells, and, as there are no sharp dividing lines, competent pathologists often differ, and so long as the separation between fibromata and sarcomata is an arbitrary one, it is often a matter of individual judgment which name should be applied. I should prefer to call the tumor of which you send a section a fibroma rather than a sarcoma, as there is so much basement substance. The fresher part of the growth shows fibrillated connective tissue, not richer in cells than many undisputed fibromata. There is an extensive and rather peculiar sclerotic or hyaline transformation going on, and enclosing dense strands and masses of cells of a fibroplastic type. I suppose it is the latter which raises the question of sarcoma; if these cells were getting the upper hand and taking possession of the field, I should incline to the diagnosis of sarcoma, but it looks to me as if the cells were being crushed out and replaced to a considerable extent by the sclerotic or hyaline tissue. I should not consider a tumor of this type likely to metastasize, although if not completely extirpated, it would be likely to recur *in loco*. The old fashioned recurrent fibroids of the surgeons often show much the appearance of your tumor."

Subsequent History—About the middle of Deecember, 1905, the girl resumed her former duties as housemaid and for a time her general bodily health was much improved, as she was now able to breathe, eat and sleep comfortably. A month after her return to work her employer informed me that since the performance of the operation her disposition seemed to have undergone a change for the worse, she being much depressed mentally, very

careless as to personal appearance and indifferent as to whether or not she gave satisfaction in her work, which formerly was not the case. This, however, was thought to be largely due to the girl's disappointment of the lack of improvement in her ability to speak distinctly. The enlargement on the right side of the tongue, previously mentioned, failed to diminish in size, as it was hoped it would do and by February 1 it had perceptibly increased in size. Hence an operative procedure for its removal was advised.

Second Operation—The patient accordingly entered my service at the City Hospital on March 12, 1906, and assisted by Dr. Gwathmey and Dr. Sherman, a second operation, similar in detail to the one already described, was performed, the incision this time, however, being made over the right side. When the mucous membrane and overlying muscular tissue had been divided and dissected back for a short distance, nine separate encapsulated tumors of various sizes were easily shelled out, the slight hemorrhage being readily controlled by the sutures necessary to close the wound.

Result—The convalescence was uneventful, the wound healing primarily. The same differences of opinion were expressed by the pathologists who examined the tumors removed at this operation, as in the first instance. Early in April, about three weeks after the performance of the last operation, a growth which grew rapidly, was noticed making its appearance in the scar tissue on the right side of the tongue. On April 12, when it was removed, it measured two by one inches in size. This growth was not encapsulated as the other tumors previously removed had been, but was intimately connected with the tongue tissues and a wide cut was made around it.

Examination of Tumor—When this tumor was submitted to the pathologists previously mentioned, three were in favor of fibrosarcoma, one was in doubt and one retained his original view as to the fibrous character of the neoplasm. Dr. F. M. Jeffries reported as follows: "The last two sets of growth removed from the tongue are identical in structure with those removed at the first operation. Even in view of the opinions expressed that this is a benign growth, I am still of the opinion that we are dealing with a sarcoma; not alone am I influenced by the fact of its frequent return, but still more so by the fact that at the last removal, this same tissue is no longer encapsulated as it was at first, but now invades and replaces the muscular tissue."

Professor Dunham wrote: "In the last specimen of tumor of the tongue received from you on May 7, I find several places, especially blood vessels, in which the tissue is much more highly cellular than in the previous specimens I have seen from the case. This makes me believe that there is a sarcomatous element in the tumor, and that there is a chance for the development of malignant qualities, though the more highly cellular parts of

this specimen are deeply situated and not at the periphery of the growth. There is also evidence in this specimen that the neighboring muscle has been infiltrated with the tumor and that the muscle fibres were undergoing degeneration and atrophy at the time the specimen was removed." In a more recent letter, Dr. Dunham says:

"The case is unique in my experience. It seems to me that your suggestion of designating the growth as a fibrosarcoma is wise."

Dr. William R. Williams, by his assistant, Dr. Krumweide, writes: "After further examination of tissue from the case of N. M., I still consider that it is morphologically fibroma. The tissue from the second operation more closely resembles that from the first, that from the third is more like fibrous tissue resulting from scar formation, although there are places which simulate the original growth. In view of the further clinical history, the diagnosis would have to be reconsidered, although the specimens themselves, to my mind, do not suggest sarcoma."

Dr. W. H. Welch writes as follows: "I have examined the sections last sent of the tumor of the tongue. I do not recall having seen in the first set of sections, the structures which I find in these later ones. There is a peculiar architectural arrangement of the tissue, characterized by bands and coils, cut variously, with nuclei along their margins. These bands and coils, which appear nearly homogeneous with a low power and staining with eosin, show with a high power a finely fibrillar and granular structure. In other places the tissue is wide-meshed, suggestive of edematous connective tissue. Here are long ribbon-shaped cells, with a peculiar longitudinal striation of the cytoplasm. It may be that these cells and bands belong to fibrillated connective tissue; in which case I should call the tumor a fibroma, but my suspicion is aroused of the possibility of a rhabdomyoma, although I can not prove it from your sections, as I do not satisfactorily make out cross-striation. Still in many rhabdomyomata longitudinal striation is the only type of striation which can be distinctly made out. Under the circumstances, it seems to me very important to apply other stains and reactions which will shed light on the nature of the fibrillation in the coils and bands referred to and in certain peculiar elongated cells found in the mucoid-like areas. Mallory's stain for connective tissue fibrils, Weigert's elastic tissue stain and Van Gieson's stains should be applied. Until the question of possible rhabdomyoma is cleared up, I should not be willing to make a positive diagnosis. There is certainly something very peculiar about some of the cells and tissue in the tumor, but the interest is pathologic rather than clinical, I think."

Dr. Horst Oertel, Director of the Russell Sage Institute of Pathology and Pathologist to the City Hospital, was now asked to examine and re-

port on the different specimens of tissue removed at the various operations from the patient's tongue, which he kindly did, and his reply is as follows:

New York, March 6, 1908.

Dr. F. H. Wiggin,
55 West Thirty-sixth Street,
New York City.

My Dear Dr. Wiggin—I have examined the tumor masses which you gave to me the other day, and also have looked over some specimens which Dr. Martland made for me, applying special stains, and can report on them as follows:

The main mass of the tumor tissue is made up of large or small coils of thick, in places almost homogeneous, masses, the ground substance of which seems to be made up of very rich protoplasmic thick fibres. Interpolated between these coils are distinct fibrillated bands. The protoplasm of these fibrillated bands shows irregular longitudinal striation. At the periphery of the fibres, and especially abundant at the periphery of the coils, are groups of nuclei which are quite characteristic in their appearance; they are long, slender, rich in chromatine, and not vesicular. A considerable number of lymphatics is rather irregularly distributed through these coils and also capillary blood-vessels. Around them there is frequently an aggregation of nuclei, similar to those previously mentioned.

In the sections which are stained according to Mallory, the circular arrangement of the more homogeneous masses and thick bands which make up the various coils appears very prominent. The main bulk of the tumor does not take Van Gieson's stain, and then only irregular bands of thin, fibrous tissue can be seen plainly traversing through these previously described cords. Weigert's elastic fibre stain shows the absence of such fibres.

In view of the arrangement of the fibres, the striation of the protoplasm in portions in which individual fibres can be made out without difficulty, the appearance of the nuclei, which in their character I consider quite typical, the fact that the main bulk of the tumor does not react to the stain for fibrous tissue and elastic fibres at all, but reacts to that of Mallory, I am satisfied that we have to do here with a myomatous growth. The derivation of the tumor, as well as its longitudinal striation, make it certain to be a rhabdomyoma, and the active proliferation of its nuclei at the periphery of the coils and around the lymph spaces, as well as the clinical features of it, stamp it, what may be spoken of, as a rhabdomyoma sarcomatodes.

These tumors, as you will recall, are exceedingly rare, and especially in the tongue. I do not recall at present how many well authenticated cases

of rhabdomyoma of the tongue have been described, but they are exceedingly few.

I shall be very much pleased to hear of the ultimate fate of the patient.

Very sincerely yours,

Horst Oertel.

Recurrence—April 19, a week following the third operation a recurrence in the line of incision was discovered which grew much more rapidly than the former one had, threatening, at the rate of progress it was making, soon to fill up the pharynx. Further attempts to remove the disease by an operation less extensive than the entire removal of the tongue seemed unwise, and even this procedure did not at this time seem to hold out much hope of the patient's permanent recovery.

Administration of Trypsin and Pancreatic Extract—An editorial in "The Journal of the American Medical Association"[1] had directed my attention to the work of Professor John Beard, lecturer in comparative embryology in the University of Edinburgh, concerning the origin and treatment of malignant disease, and as this case was now considered practically a

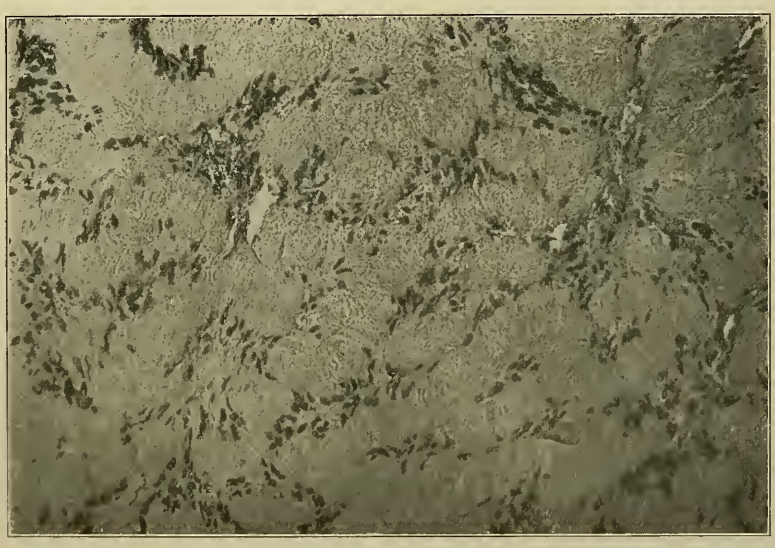

Fig. 1

PHOTOMICROGRAPH OF SECTION OF RHABDOMYOMA SARCOMATODES OF THE TONGUE MADE BY DR. HORST OERTEL AND DR. MARTLAND.

hopeless one, it was determined to test the efficacy of the treatment and some ampoules containing the trypsin preparation for hypodermic use known commercially as injectio trypsini, also some capsules containing three grains

of pancreas extract, known commercially as holadin, were obtained from the manufacturers, Fairchild Bros. and Foster of this city. Beginning about April 24, ten minims of the injectio trypsini, diluted with double its volume of sterile water, were injected into the cellular tissue of the patient's left arm over the deltoid every other day, and a capsule containing the pancreatic extract was given an hour before each meal. After about two weeks of treatment the hypodermic injections were given daily, and gradually increased until forty minims were administered daily and this treatment continued until June 15.

Result—At the time this treatment was begun, there could be felt a large growth approximately two by one inches in size at the site of the operation on the right side of the tongue, but well back, and a smaller one behind the first one. Soon after the administration of the trypsin and the pancreatic extract had been begun, it was noticed that the tumors had apparently ceased to increase in size; they remained stationary, however, until the injections were given daily and the dosage increased to fifteen minims. They then slowly began to decrease in size, but when the dosage had been increased to forty minims daily, about June 5, there was a rapid diminution in the size of the growths, especially in the larger one, which was perceptible from day to day, until when the treatment was discontinued about June 15, at the patient's request, the growths were barely perceptible to the touch and the patient was able to speak distinctly for the first time since the treatment began. During the early part of this trypsin treatment, April 24 to June 1, the patients' general health improved greatly as shown by her improved looks, her more cheerful disposition and a gain of eleven pounds in weight, and while she complained of the pain following the injections, no serious inflammatory reaction followed them and no abscess formed. Soon after the dosage had been increased to forty minims daily, the patient began to complain of severe headaches, but as she was of a pronounced hysterical temperament little attention was paid to this, beyond the giving of cathartics. The patient soon began to show signs of mental depression, became very careless of her appearance and of her work; she complained of more or less constant headache, of constipation and looked anemic. At her request, the treatment was discontinued June 15, although, as previously stated, the growths at the back of the tongue had not quite disappeared and it seemed as if only a few more doses would be needed to effect this much to be desired result. The patient continued, however, with more or less regularity to take the capsules containing the pancreatic extract until about the middle of July, when they were finally discontinued.

Administration of Amylopsin—In a communication to the "Medical Record," June 23, 1906, Dr. Beard called attention to the fact that the trypsin injections are liable to be followed by certain bad symptoms, thought to

be due not to the trypsin itself, but to the absorption of septic material from the degenerating tumor. These manifestations were stated to be nausea, vomiting, pain in the back, sleeping in any position and mental and physical torpor, and that these unpleasant symptoms could be made to disappear by the introduction into the body of diastasic ferment. Accordingly some ampoules of a solution of pancreatic diastase amylopsin, prepared for hypodermic use by Fairchild Bros. and Foster and known commercially as injectio amylopsini, were obtained and with the patient's consent, injections of this preparation were given daily, beginning with a dosage of ten minims, increased to twenty. This treatment for a time seemed to make a decided improvement in her condition, she became less morose, complained less of headache and for a time took more interest in her work. She also said that she felt better after the injections. The weather was very hot and trying at this time (July) and at her request the treatment was discontinued after only twelve ampules of the injectio amylopsi had been used. From June 15 to August 15, the neoplasms seemed to remain stationary in size, but by September 1, when the patient was next examined, it was found that the larger of the two growths had undergone a perceptible increase in size; this, the patient said, she had also noticed and was much depressed mentally thereby, but she refused further treatment and passed temporarily from under observation.

At this time she did not appear to be in good bodily health, being anemic, constipated, and having frequent headaches; much of this condition under ordinary circumstances could be fairly charged to the discomfort of remaining in the city throughout the heated term, which that year was especially trying.

On February 26, 1907, the patient reported to and was examined by Dr. George B. Hope, surgeon in chief of the Metropolitan Throat Hospital, who wrote me as follows:

Copy.

Metropolitan Throat Hospital,
351 West Thirty-fourth Street, New York.
February 27, 1907.

Dear Doctor Wiggin:

The patient operated on by you for tumor of the tongue, reported February 26, current, at the Metropolitan Throat Hospital.

The present appearance of the patient is excellent, having greatly improved in weight and color and without apparent nervous symptoms, the respiration is quiet and without effort; the voice is clear and resonant. The appetite is reported as good and the digestion regular. The tongue tumor remaining is not much more than apparent to the touch in the line of the

scar tissue on the left side and is said to be non-progressive. There is no induration surrounding, and the color is normal. There is no pain and no discomfort whatever; in fact, the patient considers herself contented and perfectly well.

The tongue was also examined by Doctors T. A. Smith and Cyrus Strong at the same date.

Very truly yours,

(Signed) G. B. Hope.

This case is of interest, aside from its extreme rarity, on account of the difficulty encountered by the pathologists in reaching a conclusion as to whether or not it should be classified as a Fibroma sarcoma, or rhabdomyoma sarcomatodes, and in connection with this subject the following statement of Virchow's in regard to fibrous tumors should be recalled: "It is a matter of great importance to remember that the boundaries of tuberous fibroma are not clearly defined from those of other tumors, especially those of sarcoma, there being undoubtedly transition forms which exist between them."

From the viewpoint of treatment, the case is also of interest. In the course of an article entitled "The Cancer Problem," Dr. Beard[2] set forth the following scientific reasons on which he bases his recommendations for the treatment of malignant diseases by trypsin: "Any ordinary cancer or sarcoma is a new development of trophoblast due to the attempt of a germ cell to start the cycle anew. It has been established that in the normal life cycle of development in any of the higher animals there are two generations, an asexual one, the larva or phorozoon, and a sexual one, the embryo; that the former was mainly, if not entirely, represented in mammalian development by the trophoblast and that in every normal development the trophoblast is suppressed by the sexual generation, its degeneration commencing at the critical period with the completion of all parts of the embryo. The change in nutrition, initiated at the critical period in vertebrate animals from fishes to man, is based in the commencing functional activity of the pancreas. This introduces an alkaline digestion by means of pancreatic juice with its various ferments. A malignant tumor, a cancer or sarcoma like the trophoblast eats and erodes its way, destroying tissues by an intracellular acid digestion." The enzyme of this intracellular acid digestion, discovered by Eugen Petry[3] in 1899, has been named "malignin" by Beard. Again, the latter states in the course of the article already quoted from: "Briefly, as I conceive it, in normal development, and in a malignant tumor the matter is simply a question of the victory of a stronger enzyme over a weaker one. The whole story is but another example of that antithetic alternation which underlies all the phenomena of living things. The solution of the problem

of the functional relation of embryo and trophoblast, how the latter nourishes itself by an intracellular acid peptic digestion and degenerates slowly by a pancreatic or alkaline digestion, becomes at the same time the embryologic if not the medical resolution of the problems of malignant disease, as well as of chorio-epithelioma."

In another article entitled "Are the Problems of Cancer Insoluble?" Beard[4] says: "Cancer is an irresponsible trophoblast or chorion, the corollary of that thesis is that under the action of injections of trypsin the cells of, say, Jensen's mouse tumor must degenerate. In the cancer ferment malignin and in trypsin we have an antithesis of ferments of which the latter is the most powerful." Acting on these views, Beard inoculated a number of mice with Jensen's mouse tumor and began the injection of two minims of a 1.5 per cent. solution of trypsin in sterilized distilled water into one of them. One of these mice died apparently from the result of an accident on the tenth day, after four of these injections had been given. The post-mortem examination revealed no cause of death, and but for the presence of the tumor the mouse seemed healthy. Microscopic examination seemed to demonstrate that every single cell of the tumor was in degeneration, fully one-half of them being represented by shapeless masses of particles, probably remains of nuclei, and all the rest were skeleton cells. The somatic tissues of the mouse, as represented by the leucocytes and connective tissue cells, were normal. The second mouse received nine injections in twenty-two days and was then killed and the tumor exposed, the growth being about the size of a lentil. The cells were all found to be degenerated. On the same day that this mouse was killed an untreated control mouse inoculated with the same tumor at the same time died of its cancer; its tumor was as large as the terminal phalanx of a man's thumb.

The publication of the results of the trypsin injections, coupled with the advice that it should be used for patients suffering from malignant disease, based largely on theoretical grounds, and before a reasonable clinical experience had been had with it to enable the investigator to determine how the trypsin should be best prepared for use, the proper dosage and duration of the treatment, and the popular articles concerning this work of Beard's, written by an injudicious friend and published in various lay journals, in my opinion, have been most unfortunate and probably would not have occurred had Beard been a physician. The result could only be prejudicial in many ways to the treatment, no matter on how solid a foundation it may rest. In the first instance, solutions of trypsin wrongly prepared, of improper strength or improperly used by injections into the tissues near the site of the growth, or used for too long or too short a time or in too large or too small doses, would surely lead to unsatisfactory results and to wrong conclusions. Since January 1, 1908, various articles and editorials in medical journals have

appeared combating the views of Dr. Beard and condemning the use of trypsin in malignant disease. Notably an article entitled "Trypsin in Maligant Growths," by Dr. W. A. Pusey,[5] of Chicago, who makes the following statement: "In one or two patients there was bitter complaint occasionally after an injection, frequent inflammatory swellings resulted and in six or eight instances abscesses developed at the site of injection." In one of his cases Pusey believes that the patient was positively benefited by the treatment with trypsin and that appreciable harm was done to others. These last had chills and Pusey believes failed more rapidly than they were failing before. Dr. Beard,[6] in a communication entitled "Trypsin and Amylopsin in Malignant Growths," makes the following statement: "The treatment has really only been in use for six months and naturally it is still in the experimental phase of its existence. The results recorded by the author (Pusey) can hardly be called good by any stretch of the imagination and did I not know of better ones I, as the scientific originator of this treatment, might well despair of it. My lips are closed regarding results, but shortly they will be forthcoming and the unfavorable cases will not be in proportion to the favorable ones as seven to one. As to abscesses, I have known as many as eighty ampoules to be given in America to a single patient with only one abscess and altogether I have as yet only heard of about six abscesses all told, excluding the record number mentioned by Dr. Pusey. Over here we (Dr. Robert Maguire, F. R. C. P., and myself), have, I think, considerably improved this treatment. The very good results obtained in certain very serious cases in Europe (to name a few, a case of cancer of the rectum, one of inoperable cancer of the tongue and two of cancer of the uterus) point to the following as on the lines of right treatment. After a few preliminary injections of small amounts (half an ampoule of the injectio trypsin) the course should be: 1. Injectio trypsini daily up to two ampoules for three or four weeks. 2. Then one ampoule of injectio trypsini on a certain day, followed by an ampoule of injectio amylopsini on the next day, and so on alternately for about four weeks. 3. Lastly, one or even two ampoules of the new injectio amylopsini daily for about four weeks, or longer, in the judgment of the physician. If using this injection any injections be still not well borne, the injection should be further diluted and given more frequently, but so that the total daily amount is not thereby diminished."

In making these injections I have found the following described technic to be satisfactory: The ampoule containing the trypsin or amylopsin is washed with 95 per cent. alcohol before it is opened. The hypodermic needle and the patient's skin at the site of the puncture are also washed with the same fluid and the wound made by the needle is sealed with collodion. Judging by this formula of treatment, it would appear that the injectio amylopsini should have been used much earlier than it was in my case and

should have been continued longer. For the failure to do this last the patient was responsible.

As has already been stated, trypsin is thought by Beard to have no harmful effect on normal body tissue, the soma or sexual product, and that whatever unpleasant symptoms arise in the course of this treatment are due to absorption of septic material from the degenerating tumor mass. The position, scientific attainments and reputation of Professor Beard entitle him to a respectful hearing, and no good reason known to me can be advanced at the present time for not carefully carrying out and further investigating this line of treatment in at least the inoperable cases of malignant disease that may come under our care, until the truth or falsity of Dr. Beard's views are fully established. For what otherwise have we at this time to offer these unfortunate people?

By presenting this case, inconclusive as it is, it has merely been my wish to lay the facts before you and to call serious attention to a most important and interesting subject, which might otherwise be overlooked, in the hope that further discussions of and clinical experience with trypsin and amylopsin within a reasonable time will demonstrate beyond question that we have at our disposal a sure and efficient remedy for the treatment of malignant disease.

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4. Medical Press and Circular, Dec. 20, 1905.
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ISOLATED TUBERCULOSIS OF THE GREAT TROCHANTER.

With a report of two cases.

By Nathan W. Green, M. D.,

Assistant Attending Surgeon.

Tuberculosis of the head of the femur, involving the hip joint, is a very familiar condition, but reports of tuberculosis of the great trochanter alone are comparatively infrequent. It may be more widely recognized than reported. It is not improbable that one or more cases have occurred in the experience of nearly every one doing general surgical work. The literature

on the subject, however, is scanty, and the condition is rarely classed separately. The importance of differentiating this condition from tuberculosis of the hip joint will be recognized by the general surgeon, and it places upon him the responsibility of its cure.

The condition in its early manifestations is difficult to differentiate from an epitrochanteric bursitis, from periostitis and from early involvement of the hip joint itself. In its latter manifestations it presents the appearance of a chronic osteomyelitis with sinus formation and with thickening in the region of the great trochanter. When these conditions exist and the patient is able to perform many of the movements of the hip joint without marked discomfort the involvement of the joint may be ruled out. The points, then, of usual tuberculous infection in the upper end of the femur being fairly well understood, it is not difficult to locate a focus in the great trochanter as opposed to one either in the upper end of the shaft or the head of the femur or in the acetabulum. When the diagnosis has once been made the method of cure will have been indicated. A thorough and wide dissection of the fistulous tract should be made tracing it down to its beginning, which will be found as a necrotic cavity in the bone where will often be located a small sequestrum. The cavity should be treated on general surgical principles, the wound packed with gauze and the whole allowed to granulate from the bottom. The resulting scar is necessarily extensive, but the healing of the wound should be accomplished in a few weeks. It is very gratifying to see the result of a case so treated.

In looking up the literature of the subject the following has been found: Pétrequin, as quoted by J. B. Petit,[1] reported in the *Gazette Medicale* for January, 1837, a case of osteitis of the great trochanter following an abscess "formed probably in the bursa over the great trochanter."

In von Bergmann and Bull's Surgery,[2] König is quoted as reporting 5 cases out of 381 cases operated upon where the focus was primary in the trochanter. There is also an illustration and description of a case of von Volkmann's.

C. G. Cumston says: "The great trochanter is frequently attacked by extra-articular tuberculosis which may develop an external or posterior abscess which makes it frequently very difficult to differentiate from coxalgia. It may be said that it is the only point of the upper end of the femur which can become invaded by tuberculosis without participation of the synovial membrane of the hip joint."

He says in regard to the diagnosis of this condition that "the significance of the signs noted in and about the joint appear to me quite impossible to describe, but personally I consider these slight secondary arthritides as merely arthritides of neighborhood because they get well easily and quickly." He offers this only as an hypothesis.

In von Bergmann and Bull's Surgery is found the following: "What it is that determines the site of the perforation of the focus is still unknown; not only the foci lying close beneath the periosteum or developing in the trochanter may perforate outward, but occasionally also those deeply situated in the neck and head. A long fistula then burrows from the head through the trochanter to the surface."

The following two cases* occurred in the surgical service of the City Hospital. Both are reported by the courtesy of Dr. Howard D. Collins, visiting surgeon, on whose service they occurred and who performed the operation on the first case.

J. H. D., 46 years of age, was admitted to the hospital October 23, 1906, with a diagnosis of tuberculous disease of the hip joint. His chief complaint was pain in the right hip and knee with a discharging sinus of the hip. The family history was negative, except for the fact that his father died of pneumonia at about 55 years of age, and two brothers died of probable pneumonia. He was by occupation a mason. Previous history: moderate drinker, has led a regular life and denies specific disease. His present illness dates from childhood. At about 12 years of age he fell from a wagon and struck on his right hip. He recovered from the immediate effects of the fall, but about two months later the hip became painful and he was unable to walk. Two or three months later an abscess in the region of the hip was opened and drained. After that he was able to get about fairly comfortably and has done so ever since. For the past six months he has had discharging sinuses. About six weeks before admission the joint became painful and he was unable to bear much weight upon it. This did not improve so he sought relief in the hospital. On the 4th of April, 1907, the old sinuses were enlarged, scraped and drained under anaesthesia by Dr. Collins.

After four months of conservative treatment with no decided improvement, he was again operated upon on the 14th of August, 1907. At this time the sinuses were more carefully explored and were found to converge at the upper and outer surface of the great trochanter. In the bone a necrotic scale 3 centimeters in diameter was easily removed and the containing cavity freely curetted. None of the sinuses led to the hip joint and it was perfectly evident that the cause of the sinuses had been eliminated. The wounds were left wide open and packed with gauze.

His highest post-operative temperature was 103 deg. F., and his pulse 128 on the day after operation. He made an uneventful recovery and on the 13th day of December, 1907, the following note was made: "All healed,

* These two cases were presented before the Society of the Alumni of St. Luke's Hospital at a meeting held during the fall of 1907.

except small area on skin." He was discharged cured and it has been impossible to locate him since leaving the hospital.

The second case was that of T. McN., aged 48. He was admitted June 25, 1907, with a diagnosis of tuberculous disease of the hip and double inguinal hernia. His chief complaint was in reference to the condition of his hip. The family history was negative. Occupation, laborer. Previous history: alcoholic. For many years had frequent attacks of cold in the chest with more or less constant cough. No tubercle bacilli were reported in the sputum. He denied syphilis. Present history: Thirty-five years ago patient fell, striking upon his hip. The hip presented a swelling, which later ruptured spontaneously, discharging pus. It then apparently healed. Ten years ago it again broke down, and he was operated upon in Bellevue Hospital. It was again healed until three weeks before admission, when his wound reopened and was again discharging. The patient was able to perform all the motions with the joint, but experienced a dull, heavy, aching pain when moving about. He lost about ten pounds in the two weeks previous to admission. On September 6, 1907, he was operated upon by myself and the fistulous tract followed to the bone, where was found a cavity admitting the end of the thumb and containing a small sequestrum. The granulation tissue about the tract was scraped and cut away and the walls of the bone cavity curetted, the whole tract being packed with iodoform gauze. His highest post-operative temperature was 101 2/5 deg. F.; his pulse 124. He was allowed to sit up fifteen days later and on November 16 was discharged improved. The pathological report was as follows: Specimens from hip of McN. show "old granulation tissue infiltrated with polynuclears and in places necrotic; no giant cells, probably tuberculous."

As he was some time in convalescing from his operation, he became rather desirous of leaving the hospital and so his double hernia was not interfered with. He was seen on February 18, 1908, at his home. The hip wound was then found healed and he was looking for work.

The points of interest in these two cases are the following: In both the diagnosis of extra-capsular tuberculosis was made before operation, although both had been admitted to the hospital as suffering with hip joint disease; in both cases isolated tuberculosis of the great trochanter was found; in both the operation was co-extensive with the fistulous tract, and in both cases the patients left the hospital with a useful and movable hip joint.

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(2) E. von Bergmann and W. T. Bull; A System of Practical Surgery, Vol. III., p. 464.

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REPORT OF THE FIRST YEAR'S WORK IN ORTHOPAEDIC SURGERY IN THE CITY HOSPITAL.

ARTHUR H. CILLEY, M. D.,

Assistant Attending Surgeon.

The orthopaedic work done by the assistant surgeon has been somewhat varied and somewhat successful, though not as varied nor successful as he could wish. The work has been chiefly the immobilization of joints with plaster of paris dressings; bloodless reduction of the deformity in cases of rigid weak foot, and an attempt at the regulation of the diet in tuberculous joint disease. There was one operation for coxa vara at which he assisted and he curetted one case of tuberculous hip joint disease with abscess. He has also recommended braces and supervised their construction in the out door department of the Hospital for the Ruptured and Crippled.

The work in plaster of paris comprised the application of spicas on cases of tuberculous disease of one or other hip, and jackets on cases of disease in the spine; the application of spicas on the case of coxa vara and on two fractures of the neck of the femur in which the Whitman method of abduction was tried; and the application of a proper dressing following the operation for rigid weak foot. There were also several cases of synovitis of the knee and osteitis of the tarsus treated with plaster. Casts of feet were made from time to time for demonstration to the house staff. The case of coxa vara was so unfortunate as to get delayed union in her right femur and still more unfortunate in having this bone broken again several months later in an attempt to break up some of the adhesions that had formed in both joints. However, a new spica was applied immediately and when it was removed in three months, union was firm and the motion was at least better than was to have been expected. Motion was much restricted before operation only about 90 deg. of flexion being possible with practically no abduction at all. After operation the left hip retained its amount of flexion, while the right hip had about 60 deg. Lateral motion was about one-third of normal in both sides. The cases of fracture of the neck of the femur, two in number, and in both the left femur gave one good and one negative result. The good result was firm union in a woman 67 years old. but the patient was lost sight of after her discharge, so that the functional result is unknown. The case with negative result has been operated on since January first, by open incision and is still in plaster of paris. Several of the cases of weak foot were so severe that one manipulation was not sufficient, but except where the foot deformity was simply part of a general arthritis the results are good and the patients either at work at the hospital or discharged cured. These patients were given braces at the out door department

of the Hospital for Ruptured and Crippled, as were several of the hospital helpers, whose feet were not in bad enough condition to require operation. Braces were also made for one case of drop foot following a successful operation for Jacksonian epilepsy by Dr. Dawbarn, and one case of anterior poliomyelitis due to potassium cyanide poisoning under Dr. Joseph Collins, Visiting Physician.

There have been only three cases in which any attempt has been made to regulate the feeding in tuberculous disease of the bones and joints. All were in desperate condition and none has been cured. All were and still are in Male 9. One was operated on first for left inguinal adenitis. The pus either came from the retroperitoneal glands or the disease extended upward to them requiring later operations above the rim of the pelvis with counter opening in the ischio-rectal fossa. Later his left hip was excised and when his feeding was started his condition was miserable in the extreme. There was profuse discharge from sinuses in the groin, anterior thigh region, perineum and around the greater trochanter. The dressings which were voluminous required changing every day. Now his morale is good—he gets about with crutches—he is gaining weight steadily, and there are only two sinuses left with discharge sufficient to stain the gauze in two or three days.

The second is also a left hip. He had been extensively operated on prior to his admission to the City Hospital and had three large drainage tubes and several gauze strips in as many sinuses. He also required daily dressing as he was apparently "running away to pus," and he was a bed case as he had been ever since admission. He was interesting at first on account of his flat refusal to follow the prescribed regimen. He was told that he could try to get well or not as he chose and when he saw the beginning good result in the first case he quickly fell in line. He is also interesting on account of two attacks of erysipelas, each following the application of plaster of paris to his left foot, which is in marked equino-varus. This deformity is probably due either to trauma to the sciatic nerve during his extensive operations or to pressure on the nerve from the infiltration about the hip joint and inside the pelvis. It is unnecessary to say that no further attempt has been made to correct his deformity, but, nevertheless, he has had a third attack of erysipelas. He is now out of bed going around in a wheel chair, his tubes and packings are all out, his dressing is changed two or three times a week.

The third case, right hip disease, is the one operated on in the City Hospital by the writer. He had at the time of operation a discharging sinus under about the middle of Poupart's ligament and a large abscess extending around the greater trochanter, buttock and down the anterior and outer aspects of the thigh nearly half way to the knee. The abscess was

freely opened, the trochanter found diseased and curetted almost entirely away. He later developed a superficial abscess in the left buttock. He is not doing well.

In these cases the attempt was made to carry out some of the recommendations of Dr. John F. Russell in the treatment of pulmonary tuberculosis, following along the same lines as those laid down in a paper by the writer published in the Post-Graduate October, 1906.

In spite of the present poor condition of case three the results are encouraging. The obstacles to the successful carrying out of this treatment have been many. The work is new. The possibilities of varying the food supply are limited. But worst of all is the necessity of keeping the acute and chronic cases in the same ward.

Tuberculous joint disease is a chronic condition and is not amenable to any single surgical operation. These patients require care for a long period of time whether operated upon or not. When chronic and acute cases are mixed in the same ward one suffers, either the acute cases occupy the time, thought and attention of the visiting, house and nursing staffs to the exclusion of " the chronics," or else " the chronics," as it were, get on the nerves of all concerned and the entire service is handicapped. In tuberculous disease of the bones and joints we have not a local disease, but a general disease with local symptoms. To consider these patients from the standpoint of the joint alone seems to me no more sensible than to consider a mucus patch a disease of the mucus membrane alone. The location of the lesion is more or less accidental as tuberculosis, like syphilis, may attack practically any tissue of the human body. To treat a tubercular joint as a separate thing by itself is no more sensible than to treat the mucus patch of syphilis by itself. Not so many years ago syphilis was treated in this slipshod manner. The local lesions were treated and " cured " and then treatment was suspended until a fresh outbreak showed itself. Advanced hopeless, helpless cases of brain syphilis were fairly common in those days instead of being correspondingly uncommon, now that the disease itself and not simply its local manifestations are treated.

The same thing holds true for that vast army of " chronics," the " rheumatic " or " rheumatoid " cases using these old words to include the whole army without attempting to classify them and knowing that the words themselves have no scientific meaning.

These patients should be treated where their care will be the sole occupation of their attendants. So much depends on the quantity of food that they actually eat as well as the character of it that they should be given more attention than they can be given in the general wards of any hospital. It may be said that the City Hospital is for acute cases and not for " chronics." It seems to the writer, however, that there is such a crying need for better

care of this class of patients trying to prevent the enormous expense that they occasion on account of their inability to work, that something must be done and the City Hospital is in the ideal location for this work. There is abundant land, fresh air, sunlight. Furthermore, the house staffs and the nurses have been much interested in the work and much credit is due them for the results we have.

In having a separate place for these unfortunate chronic joints all of which require more or less local attention in the way of protection, manipulation or operation, in other words, more or less orthopaedic care, in addition to their general treatment there need be no clashing of interests, but at present the scattering of these patients all over the great building makes it impossible for one man to see the various patients, leaving out his attempting any systematic treatment of them. And unlike the possibilities of brilliant surgery in many fields our results depend on persistence more than on genius.

If at some time it shall seem wise to separate our patients and give these chronic joints the chance they need, I feel sure that the results will far surpass any yet attained anywhere.

DOES FRACTURE OF THE TIBIA ALWAYS PREVENT WALKING?

Edward Milton Foote, M. D.,

Attending Surgeon.

Fracture of the lower end of the tibia due to direct violence is sufficiently rare to present interest. Two such patients have recently been under my care at the City Hospital. Both of them were able to walk almost immediately after the accident, and continued to do so, with little pain and a slight limp. About the same time there came into the hospital, under Dr. Wiggin's care, a man who had fractured both malleoli, and who was also able to walk fairly well within a few days of his injury. In all of these cases the signs of fracture were so ill-defined that the diagnosis was in doubt until a radiograph was taken. It seems, therefore, worth while to consider how far walking is a contraindication of fracture of the tibia after injury about the ankle.

I think it is generally assumed that if the patient is able to walk, and there are no positive signs of fracture of the tibia, such lesion is ruled out. Such has been my belief, and I have taught this for years to students. I have usually been able to demonstrate, once or twice a year, the ability of a patient to walk after fracture of the lower end of the fibula, but as far

as I know I have never seen a patient do so whose tibia has been recently broken. In view of the three cases herewith reported it is necessary to revise this belief.

It is only fair to say that a few authors have recognized the possibility of a patient's walking after fracture of the lower end of the tibia. Thus Leser, writing in 1890 of fracture of one or both malleoli, says: "It is not safe to assume that the function of the extremity is entirely lost. On the contrary, in many cases the patients attempt to walk and succeed in doing so with considerable pain." Helferich (translated edition), in 1902, says: "Individuals with unusual grit, sometimes even children, may be able to limp along quite a distance." This is hardly a description of the three patients who are the subject of this paper, as they walked carefully, with shortened steps; but no grit was required, as they had almost no pain.

Beck, writing in 1900, more nearly describes these patients when he says: "If the fracture extends over only a small portion of the malleoli the function of the leg may be so little disturbed that the patient is able to walk considerable distances."

Stimson also makes, in 1907, a direct statement in regard to fracture of the lower end of the tibia: "Pressure upward against the heel is not painful, and the patient can sometimes walk if he steps carefully and without much movement at the ankle joint."

These are the only references to the subject that I found in a short search in the library of the N. Y. Academy of Medicine. Ten other writers[1] either make no mention of fracture of the internal malleolus apart from other injuries, or, if they do refer to it, they say nothing as to the loss of function which follows it.

The clinical histories of the three patients are briefly as follows:

Case I.—P. P., male, aged 41, healthy. January 16, 1908, a large beam fell six feet and struck his left ankle. He was unable to rise on account of pain and was taken to Bellevue Hospital, where a splint was applied. The patient was then transferred to the City Hospital.

Twelve days after the injury there was some ecchymosis of both sides of the leg; no difference in measurements; very little swelling; no abnormal bony motion nor crepitus; but a little tenderness on pressure. Passive and active motions were preserved, and the patient walked with the gait supposed to be characteristic of fracture of the fibula—advancing the injured limb well beyond the sound one, then bringing the sound foot only

[1]These authors with the dates of publication are as follows: American Textbook of Surgery, 1903; Cheyne and Burghardt, 1900; Hopkins, 1900; International Textbook of Surgery, 1902; Pick, 1899; Rose and Carless, 1905; Scudder, 1906; Tillman (translation), 1898; Von Bergmann's System of Surgery (translation), 1904; Wyeth, 1898.

up to the injured one, never beyond it. For this reason and on account of the presence of ecchymosis after twelve days, I exhibited this patient to my students as having a probable fracture without displacement of the external malleolus. The radiograph, Fig. 1, shows that the external malleolus was intact but the internal malleolus was broken.

FIG. 1.—FRACTURE OF THE INTERNAL MALLEOLUS DUE TO DIRECT VIOLENCE.

Case II.—N. P., male, aged 32, healthy. February 5, 1908, a wagon wheel ran across his right foot. There was pain in the internal malleolus, but very slight disability in walking. Ten days after the injury there was tenderness over the internal malleolus, a false point of motion, and crepitus.

Ecchymosis extended half way up the leg. The diagnosis of fracture of the internal malleolus was confirmed by the radiograph (Fig. 2). The gait of this patient was scarcely affected. Union was perfect in four weeks.

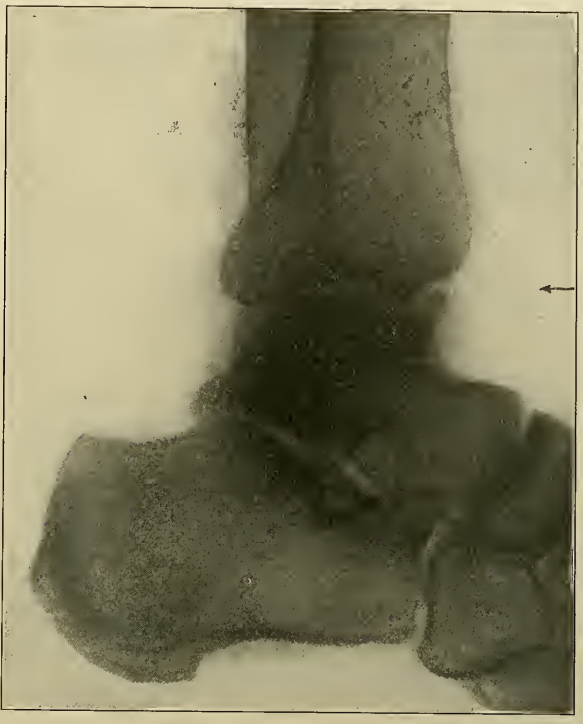


FIG. 2—FRACTURE OF THE INTERNAL MALLEOLUS DUE TO DIRECT VIOLENCE.

Case III.—J. M., male, aged 45, healthy, slipped on the ice February 15, 1908, and twisted his left ankle. He walked about a good deal, but the following day, on account of the pain, swelling and discoloration in the ankle, he went to Bellevue Hospital, where he was told he had a sprained ankle. He was transferred to Dr. Wiggin's service at the City Hospital.

Four days after the injury there was extensive ecchymosis of the whole leg and dorsum of the foot; considerable swelling, slight tenderness on pressure on both malleoli, no abnormal mobility, no pain on passive motion, but some pain on walking. The radiograph (Fig. 3) shows fracture without displacement of both tibia and fibula.

In conclusion, then, the positive signs of fracture of the internal malleolus are ecchymosis, deep swelling or thickening of the malleolus, tenderness on direct pressure, and partial loss of function, not necessarily

FIG. 3.—FRACTURE OF BOTH MALLEOLI DUE TO INDIRECT VIOLENCE.

sufficient to prevent walking. In the severer cases there will also be abnormal motion, crepitus, pain on manipulation or use, and inability to bear the weight on the foot. The value of radiographs in two planes as positively establishing the diagnosis is evident.

III.—Gynaecological and Obstetrical Divisions

100 CONSECUTIVE CELIOTOMIES: MORTALITY 2 PER CENT.

By Charles Gardner Child, Jr., M. D.,

Attending Gynecologist.

The cases herewith reported from the Gynecological Division are not selected ones, but are taken consecutively as they came to the surgical service of the City Hospital. Many of the pathological conditions met with were of such rarity that it is to be regretted they cannot be described in full. Two of the cases were absolutely unique. The different sources from which the patients were received is as follows:

Genito Urinary Division	41
Admitting office, Twenty-sixth street	22
First Medical Division	8
Second Medical Division	5
Surgical Division	5
Maternity Division	3
Dermatological Division	3
Opthalmological Division	2
Neurological Division	2
Bellevue Hospital	2
Admitted direct	7

The operations were all performed by me personally, or under my direct supervision, and gas-ether anesthesia always employed unless contraindicated. The after treatment was made as simple as possible, regular diet being allowed as soon as the ether nausea had subsided, and the patients allowed out of bed on the tenth to fourteenth day.

Age:

Youngest	16
Oldest	56

Average age 27.1 years.

Menstruation:

Earliest at	10
Latest at	20

Average age at onset 14.4 years.

Two cases began menstruating at the early age of ten years, both in Ireland. One case, a native of the United States, sixteen years old at time of operation, had never menstruated.

Average duration 4.6 days.

Regular in	68 per cent.
Irregular in	31 per cent.
Scanty in	14 per cent.
Moderate	36 per cent.
Profuse	49 per cent.
Dysmenorrhea in	63 per cent.

Fifty-one of the cases were multipara who had born 136 children with 38 miscarriages. The rate of sterility was 26 per cent.

The following pathological conditions of the pelvic organs were met with:

Uterus:

Retrodisplacements	40
Procidentia	4
Lateral deviations	3
General fibrosis	7
Fibromyoma	4
General fibrosis and multiple fibroids	1
Fibromyoma and carcinoma of the cervix	1

The retrodisplacements were corrected by shortening the round ligaments, the vaginal route being used eight times and the abdominal twenty-six. Ventral suspension was performed twice, ventral fixation once and vaginal fixation once. In two cases the uterus, when freed from adhesions and restored to position, showed no tendency to retroversion and the ligaments were not shortened. For the lateral deviations the round ligament of the opposite side only was shortened.

The cases of procidentia were treated by shortening the round and uterosacral ligaments twice, the round and infundibulo-pelvic ligaments once, and in one case the round and broad ligaments were shortened and ventral suspension performed. In all four cases the relaxation of the vaginal outlets was corrected by Emmet's operation.

The uterus was removed twenty-eight times by the abdominal route. Supravaginal hysterectomy was performed six times for general fibrosis, four times for multiple fibroids, once for general fibrosis with accompanying mutiple fibroids, and in sixteen cases of pyosalpinx. Panhysterectomy was performed twice for general fibrosis and in one case of tubo-ovarian abscess.

TUBES:

Chronic salpingitis	65
Pyosalpinx	25
Pregnancy	1
Tubo-ovarian abscess	14

Resection was praticed twelve times in chonic salpingitis, three times in pyosalpinx, and once in tubo-ovarian abscess.

The tube was removed fifty-three times for chronic salpingitis, twenty-two times for pyosalpinx, thirteen times for tubo-ovarian abscess and once for tubal pregnancy.

OVARIES:

Cystic degeneration	61
Ovarian cyst	3
Prolapse	12
Corpus luteum cyst	3

Resection was carried out eight times and removal fifty-six times. A right ovarian cyst had one twist in the pedicle. The largest corpus luteum cyst measured three inches in diameter and the smallest two inches. All three were removed without sacrificing the ovary. The ovarian ligament was shortened eleven times for prolapse of the ovary.

THE APPENDIX:

Adherent in	25.2 per cent.
Adherent to right appendages in	10.9 per cent.
Adherent posterior in	14.3 per cent.

In twenty-one cases it was ligated and removed, and in twelve it was inverted.

CASES OF SPECIAL INTEREST.

Case VI.—Abdominal tumor more prominent on right side and reaching two inches above umbilicus. Only slightly movable, and tender on pressure. First noticed by patient three months ago, and since then has been rapidly increasing in size with several attacks of acute local peritonitis; T. 100, P. 92.

Operation—Median line incision, multilocular ovarian cyst with one right to left twist in pedicle removed; acute local peritonitis, many old and recent adhesions.

Case XVIII.—The entire uterine cavity was filled by a large breast shaped fibroid growing from the fundus and posterior wall, the cervical portion or nipple being firmly held by the internal os and presenting at the partially dilated external os a mass of soft necrotic tissue. Left pyosalpinx with cystic degeneration of the ovary and large cyst of morgagni 2½ inches in diameter.

No. 1.—CASE No. XVIII

Case XIX.—A post operative abdomino-vaginal sinus running an unusual course. The sinus one-half inch in diameter, began in the median line, at the centre of a large ventral hernia, ran directly across to the left internal inguinal ring where it was densely adherent to the external iliac artery, vein and a coil of the iliac colon with which it communicated. From this point it ran forward, communicating with the bladder, thence down along the posterior face of the uterus, opening into the vagina at the cul-de-sac of Douglas. The sinus was dissected out entire, measuring twelve inches, and the openings in the intestine, bladder and vagina closed with purse-string sutures. Perfect recovery.

Case LXI.—Double tubo-ovarian abscess. The appendix was firmly adherent by its tip to the abscess on the right side with which it communicated by a minute opening. The pus from this abscess contained colon bacilli in plenty, none were found in the abscess on the left side. Appendectomy, supra-vaginal removal of uterus and appendages. Uneventful convalescence.

Case LXXXI.—Wandering spleen adherent in pelvis. The patient had had a panhysterectomy for squamous cell carcinoma of the cervix performed

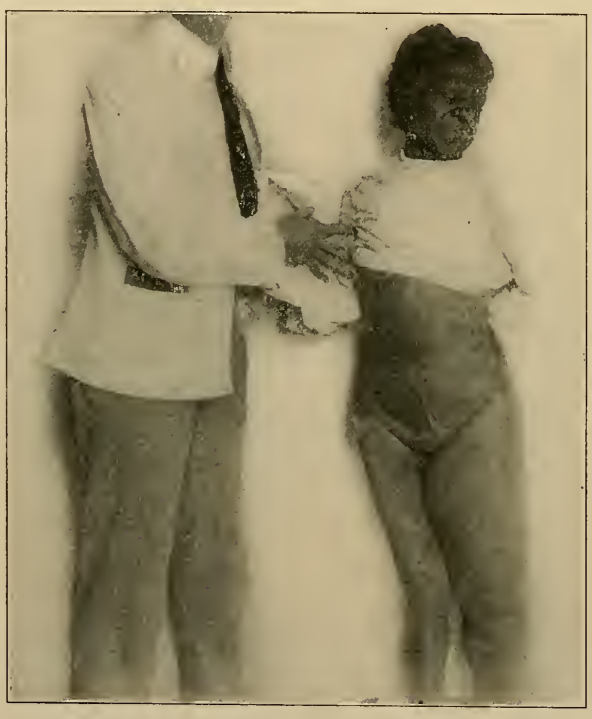
two years previously, and had suffered from vague abdomino-pelvic symptoms, increasing in severity for six months. Examination showed an adherent mass in the left side of the pelvis which was diagnosed as a recurrence of the malignant growth. A median line incision revealed an enlarged, congested spleen four inches in diameter with a pedicle nine inches long, adherent to the left broad ligament stump. The spleen was freed, a second abdominal incision made on the left side, just at the free border of the ribs, and the spleen brought through the peritoneal incision and enclosed in a pocket made for its reception between the peritoneum and fascia. The peritoneum was then closed, all but a small opening for the transmission of the splenic vessels and the fascia united over it. One year later the patient had been free from symptoms, the spleen had decreased in size and remained in its new position just at the free border of the ribs.

No. 2.—CASE LXXVIII.—CONGENITAL ABSENCE OF THE RIGHT APPENDAGES.

Case LXXVIII.—Congenital absence of the right appendages. The patient, twenty-one years of age, had begun menstruating when sixteen years old, always irregular, the flow lasting six days, profuse, and accompanied by considerable pain. For seven months previous to operation, and dating from a six-weeks' miscarriage, she had suffered from severe and continuous pain in the left iliac region.

Operation—Transverse abdominal incision. Left pyosalpinx and cystic ovary two inches in diameter. The right tube and ovary were absent, the fundus, round ligament and broad ligament being normally formed. Supravaginal removal of uterus and left appendages. Convalescence uneventful. Congenital absence of the tube and ovary unassociated with defective development of the uterus and vagina is of rare occurrence. But one case, that of Colomiatti, is reported, the right tube and ovary being likewise absent.

Case LXIX.—At operation for a retro-displaced uterus the gall bladder was found enlarged, drawn strongly to the left side and adherent to the anterior abdominal wall one inch to the left of the median line by a firm band of adhesions two inches long and one inch wide. This was cut between ligatures, allowing the gall bladder to return to its normal position without tension, and the round ligaments shortened to correct the retroversion. No history of a previous gall bladder inflammation was obtainable. The patient was relieved of all symptoms when seen at the end of six months.



No. 3.—CASE XXIV.—SHOWING ORDEMA OF THE LEFT LEG RESULTING FROM THE PRESSURE OF THE TUMOR IN THE PELVIS.

Case LXX.—Tubal twin pregnancy (See fatal cases).

Two cases out of the hundred died.

Case XXIV.—Fibromyoma of the uterus with carcinoma of the crevix. A median line abdominal incision was made, the condition diagnosed as inoperable and the wound closed. Sudden failure of both heart and respiration with death occurred as the patient was leaving the table.

Case LXX.—Tubal twin pregnancy. Mrs. G., aged 35, Vi-para, was last delivered in 1902 of twins by a protracted and difficult labor at term, followed by a short and uneventful puerperium. Her last normal menstruation was on May 20. During the month of June she had several slight hemorrhages

No. 4. CASE XXIV.—FIBROMYOMA OF THE UTERUS WITH CARCINOMA OF THE CURIX.

from the uterus, and more or less continuous pelvic pain on the left side. On the evening of July 3 severe pelvic pain, accompanied by nausea and vomiting, suddenly developed, and from this time on her condition steadily progressed from bad to worse.

Examination—I first saw her at 4 o'clock on the afternoon of July 5, one-half hour after admission, in an extremely precarious condition; temperature 105 F., pulse 142, respirations 36, and markedly exsanguinated. The abdomen was slightly distended and very sensitive to even the slightest pressure. Muscular rigidity was marked on the left side, with dullness extending from the pelvic brim to the free border of the ribs. The breasts

showed marked areolar changes, and the presence of secretion. The uterus lay in an anterior position, was slightly enlarged, and its mobility restricted. Examination of the right appendages was negative. The cul-de-sac of Douglas was filled by a soft, semi-fluctuating mass extending well out into the left fornix. In consultation with Dr. William R. Williams, a diagnosis of ruptured ectopic gestation with the presence of active bleeding was made, and immediate operation decided upon.

Operation—Through a median line incision the abdominal and pelvic cavities were found filled with a large amount of fluid and clotted blood; the left tube was ruptured in three places, one segment two inches in diameter lying free in the blood clot. Twin fetuses, of equal size, lay outside of the tube, one attached by its umbilical cord, the other free. The proximal end of the tube was bleeding profusely, the distal end was densely adherent to the ovary, evidently the result of an old salpingitis. The tube and ovary were ligated and removed. The right tube was intact and adherent to the ovary and posterior face of the broad ligament. The peritoneal cavity was cleared of blood and clots, the wound closed and the patient infused. Exitus twelve hours later with gradually rising temperature and pulse.

In the specimen removed two points of special interest were noted. The twins were of equal size, measuring 3½ inches in length, and possessed but one amniotic sac.

But five cases of tubal twin pregnancy, where one tube contains both ova, as distinguished from twin tubal pregnancy, where each tube contains an ovum, are on record.

Schauta—Two sacs of unequal size were present, one ¾ inch in diameter and the other 1½ inches. The larger fetus measured 2¼ inches in length.

Saniter—The two ova were of unequal size, the smaller ¾ inch in diameter and the larger contained a fetus 1½ inches long.

Cameron—Two amniotic sacs neither of which were measured or opened, but the estimated period of gestation was seven weeks.

McCann—One amniotic sac measuring 1¼ inches in diameter contained both fetuses, the estimated period of gestation was 2½ months. In these four cases there was bleeding from the tube at time of operation, but the amniotic sacs were unruptured. In the fifth case, that of Henricius and Kolster, the specimen was found post mortem, in a patient dying of phthisis. Two sacs were present, one containing a well-preserved fetus 51 cm. long, and the other macerated fetal remains.

ANTE-OPERATIVE TREATMENT—All cases were prepared abdominally and vaginally the day previous to operation, the vagina, external genitalia, and abdomen being scrubbed with the tincture of green soap, a poultice of which was then applied to the abdomen and external genitalia. This was removed on the operating table and followed by a final cleansing with the green soap, alcohol and ether.

Operative Technique—In the abdominal sections, when the longitudinal incision was used, the sheath of the rectus muscle, generally the right, was opened, and the muscle retracted to the outer side. At the completion of the work within the peritoneal cavity the peritoneum was closed with a continuous stitch, the rectus muscle allowed to return to the median line, and the incision in its sheath, when short, closed by a continuous suture and when more than three inches long by interrupted sutures. No sutures were passed through the fat, and the skin flaps were approximated by a subcuticular stitch of silkworm gut, removed on the tenth day. When the transverse incision was employed the original technique of Pfannenstiel was modified to some extent. In cases where the disease extended well up out of the pelvis into the abdomen the site of election was just above the pubic hair line at the transverse fold instead of at the hair line, the situation selected when the disease was limited to the pelvis. After the transverse division of the skin, subcuticular tissues and rectal sheaths, instead of incising the linea alba, the right rectus muscle, after separation from the pyramidalis was retracted outward and the peritoneum incised vertically. The self-retaining retractor of Doyen was then introduced and retained until the intra-abdominal work was completed.

While the intra-abdominal work was in progress the Trendelenburg position was invariably used, the table being elevated to an angle of 45 degrees, and only lowered for treatment of the appendix and before closing the peritoneum. The field of operation was walled off by gauze pads wrung out in hot saline solution, and in all cases of pyosalpinx and tubo-ovarian abscess the pus was removed by aspiration, when practical, before the separation of adhesions was begun, a most important point and one often neglected. When the removal of a tube was called for its deep dissection from the cornu of the uterus was practiced in most cases. In the tubal resections as much of the tube was removed as thought necessary, a dorsal opening one inch long made into the lumen of the remainder at its distal end and the serous and mucous surfaces united with interrupted sutures in order to preserve a patent opening.

In the vaginal section cases the anterior vaginal wall was incised transversely at its reflection from the cervix and longitudinally to within one inch of the urethral meatus, the bladder separated from the uterus and anterior vaginal wall, and the peritoneum divided at its uterine reflection.

Through this opening the fundus was delivered into the vagina and the treatment indicated carried out. When this was finished the fundus was replaced and the longitudinal incision in the anterior vaginal wall closed by interrupted sutures; the transverse incision was left open to afford free drainage from the wound area, from which there is always considerable oozing, and so that the anterior vaginal wall, in the retrodisplacement cases, might form its new attachment at a more desirable point higher up on the cervix.

Hemostasis was secured by the use of the Child angiotribe, and by individual ligation of the vessels. No case of secondary hemorrhage occurred.

Drainage through the abdominal wound was never employed, and through the vaginal only three times; in two cases of tubo-ovarian abscess and in one panhysterectomy.

In approaching the field of operation, the abdominal route was selected ninety-one times and the vaginal nine times. In five cases the combined abdominal and vaginal routes were employed as being better adapted to meet the conditions.

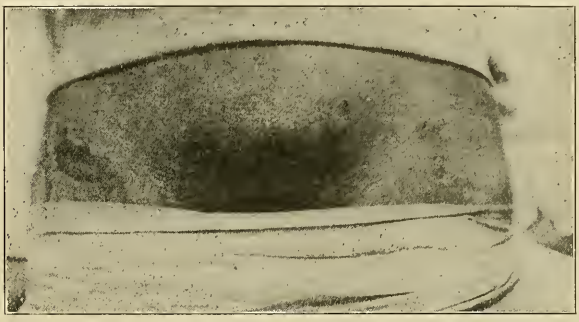
Abdominal Incision.

The transverse incision was used fifty-seven times, and the median line incision thirty-four times. In twenty-three cases the wound was closed with continuous sutures of chromic gut in the peritoneum and fascia and a subcuticular stitch of silkworm gut in the skin. The poor results obtained with the catgut led to its early abandonment and in the next thirty-six cases the peritoneum was closed with a continuous suture of fine silk, the fascia with interrupted sutures of medium size silk, and the skin with a subcuticular silkworm stitch. This gave excellent primary results, but in a number of cases the secondary results were not so good, some of the fascial sutures being subsequently discharged from the wound after several months had intervened, and in the remaining thirty cases silkworm gut was substituted for the silk and the wound closed by the following technic:

The peritoneum is brought together by a continuous suture of fine silk. The fascia is united by a running quilted stitch of medium-sized silkworm gut, drawing the raw edges together without undue tension, and instead of being tied the ends are brought out through the skin and left long near the angles of the wound. (Fig. 1, Illustration No. 6.)

The incision in the skin is closed by a continuous subcuticular stitch of silkworm gut, and the ends are also brought out long through the skin near the angles of the wound, but on the opposite sides from the fascial stitch. (Fig. 2, Illustration No. 6.)

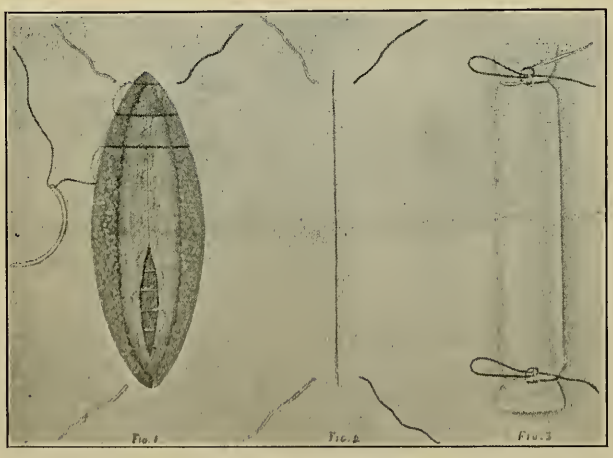
A firm roll of gauze, one inch thick and slightly longer than the wound, is now laid over it, and over this, at each end, the skin and fascial stitches are



No. 5. TRANSVER INCISION TEN DAYS AFTER OPERATION.

tied together in a single bow knot, just tight enough to take up any slipping that may have occurred in the fascial stitch since its introduction (Fig. 3). At the first dressing on the second day the gauze between these knots is cut out so as to allow subsequent daily inspection of the wound.

At the end of the first week the knots are untied, releasing the remaining ends of the gauze roller and the skin stitch is cut off at one end and drawn out. One week later the fascial stitch is removed in a similar manner, thus leaving no suture material behind in the wound between the peritoneum and skin.



In case there is failure on the part of the wound to heal by primary union the skin stitch should be withdrawn and the fascial incision inspected.

If this shows infection or if the infection is under the fascia, its edges can be readily separated by loosening the suture without removing it, and proper drainage secured. Later, when the infection has subsided and union begun the edges of the fascia can again be drawn together and good approximation obtained. Thus, the introduction of secondary sutures to close the separation in the fascia so often observed following suppurating wounds, and such a frequent cause of postoperative hernia, is avoided, saving much annoyance to both patient and surgeon.

A comparative study of the results obtained by these different methods of closing the abdominal incision is of interest.

Primary union, considered as occurring in those cases only where there was not the slightest interference with the reparative process in the wound, was obtained in 85.3 per cent. of the cases, 28.2 per cent. being septic at time of operation.

Series I.—Chromicized Catgut:

Percentage of primary unions in clean cases, 70.5.
Percentage of primary unions in septic cases, 50.
Average of primary unions, 60.2 per cent.
Percentage of cases septic at time of operation, 26.1.

Series II.—Silk and Silkworm Gut:

Percentage of primary unions in clean cases, 91.2.
Percentage of primary unions in septic cases, 95.
Average of primary unions, 93.1 per cent.
Percentage of cases septic at time of operation, 29.8.

The catgut not only gave a low percentage of primary unions in the clean cases, but in the septic cases yielded one infection to every two cases operated upon. In marked contrast were the excellent results secured with the silk and silkworm gut in both clean and septic cases, an average of 93.1 per cent. in a series of cases where 29.8 per cent. were septic at time of operation.

IV.—Division of Genito-Urinary Surgery

GONORRHOEAL RHEUMATISM, CURED BY SEMINAL VESICULOTOMY.

By Eugene Fuller, M.D.

Attending Surgeon, Genito-Urinary Division.

In December, 1904, I originated the idea of trying to cure gonorrhoeal rheumatism in the male through the employment of the operation I had devised of seminal vesiculotomy and the first gonorrhoeal rheumatic patient I operated upon was at the City Hospital in December, 1904. The result in the first and experimental case was a brilliant success. In February, 1905, I operated upon my second City Hospital case of gonorrhoeal rheumatism, the result being a complete cure.

In an article entitled "The Relation of Gonorrhoeal Rheumatism to Seminal Vesiculitis and Its Cure by Seminal Vesiculotomy," published in the "Annals of Surgery," June, 1905, these two City Hospital cases were reported together with two other cases from my service at the Post-Graduate Hospital, making in all four cures out of the four cases so far operated upon. In explanation of my early study of this subject I will quote my opening remarks in the article just alluded to in the "Annals of Surgery" and from that article also I will abstract the histories of the two City Hospital cases of gonorrhoeal rheumatism, those cases appearing in that article as Case I and Case III. The quotation is as follows:

Gonorrhoeal rheumatism has hitherto represented one of the most unwelcome complications, I think I am safe in stating, that the genito-urinary surgeon has had to encounter. In using the term unwelcome, I do not refer particularly to the element of danger, but more especially to those of rebelliousness and uncertainty. The complication has, as a rule, resolved itself when it got, as it were, good and ready. No drugs or therapeutic measures have had any markedly beneficial effect, and many are the surgeons who have in connection with these cases either lost standing by being foolish enough to definitely prognosticate the length of time the complication would in a given instance persist, or have lost patients through apparent failure of the varied measures adopted for the purpose of cure. Gonorrhoeal rheumatism is a systematic infection, the source of which is genito-urinary. It apparently bears no connection at all to acute inflammatory or to muscular rheumatism. The systemic infection causing gonorrhoeal rheumatism is generally considered to be one mixed in character. Just what rôle the gonococcus plays in this connection is still a subject open to discussion. Some are inclined to credit it with being the chief factor, and in substantiation of their contention point to the fact that that germ has been found in

affected joints and bursæ; while others assert that the only rôle played by it is a somewhat minor one. These latter contend that the gonococcus, through the localized inflammatory reaction which arises from its presence in connection with the mucous membrane, allows other germs to enter the system, the result being the usual group of septic symptoms classed under the clinical heading, gonorrhoeal rheumatism. I am personally inclined to assign myself to this latter group of thinkers, but have recently digressed somewhat even from them, for I now hold that gonorrhoeal rheumatism so called can exist entirely independently of the gonococcus; in other words, I am now positive in my opinion that another agency can exist which can play the minor role assigned to the gonococcus of allowing other germs to enter the system from a genito-urinary source. In substantiation of this personal opinion, I have observed on a considerable number of occasions all the clinical symptoms of gonorrhoeal rheumatism develop subsequent to surgical instrumentation or treatment which has awakened an acute inflammatory reaction in connection with old quiescent lesions of the seminal vesicles. I will, however, have to admit that in the cases where I have observed this resulting complication there has been an early history of gonorrhoea to perhaps account for the existing lesions in the seminal vesicles; still, the clinical and histological evidence has gone to prove that a complete elimination of the gonococcus had already and probably long before occurred.

A striking peculiarity of gonorrhoeal rheumatism is the marked susceptibility to it shown by the individuals it does attack. In other words, if one develops gonorrhoeal rheumatism in connection with his first attack of that disease, it is extremely likely that he will redevelop it not only with all subsequent similar infections, but also many times in connection with inflammatory exacerbations originating from gonorrhoeal lesions. On this account it may be that some agency aside from the element of mixed infection must be present in order to account for the development of this complication. It is very unusual for gonorrhoeal rheumatism to develop in the early stage of a fresh infection.

As it was but five months ago, namely, in December, 1904, that the idea occurred to me to try to cure gonorrhoeal rheumatism by the operative method I am now reporting, it is only since then that I have been actively investigating these cases from the stand-point of the male subject; and my special clinical study has been to try to determine whether the systemic infection in these cases seemed to enter from any special focus, or from the general mucous surface. I am as a result strongly of the opinion that the systemic infection in the male usually enters from a special focus, and that that special focus is represented by a seminal vesiculitis. In support of this assertion, I have investigated fifteen cases of gonorrhoeal rheumatism in the male. Six of these cases are from my private practice and nine

from my practice at the City and Post-Graduate Hospitals. In twelve of these the only existing genito-urinary lesion was in the seminal vesicles, namely, seminal vesiculitis. In one there was a marked seminal vesiculitis and a subsiding inflammation of the mucous urethral surface. In two, although the seminal vesicles were not free from some involvement, still, the urethral inflammation seemed to be the chief feature. To further prove my contention, I have been able to subject to my operation of seminal vesiculotomy four out of the twelve cases showing seminal vesiculitis to be the only existing genito-urinary lesion. By means of this surgical procedure, all systemic absorption from the seminal vesiculitis has been immediately checked and a resolution of the genital lesion has promptly followed. Almost immediately in all these cases the active symptoms of gonorrhoeal rheumatism have wholly disappeared. Firm manipulation of the parts previously affected has been possible at the end of twenty-four to forty-eight hours without provoking other than normal sensations, and movements of the joints in bed has caused no pain. At the end of two weeks, when the patients have been allowed out of bed—that being the usual period of rest demanded by the operation—they have all been able to walk about, and to leave the hospital promptly, entirely free from their gonorrhoeal rheumatism. As two of these cases had been bedridden for four months, and one of them for three months previous to operation, considerable atrophy had taken place in the muscular structures of their thighs and legs, and their joints, from long disuse, had become somewhat stiffened. It was consequently natural, when these individuals were put on their feet two weeks after operation and made to walk, that they should at first have found themselves weak and awkward, and that their ankles after they had been up some time would for the first few days have swelled somewhat. All of them were fearful, when they first began to walk, lest the symptoms just described due to non-use of their limbs were not indicative of a return of their gonorrhoeal rheumatism. As each day they walked better and felt better, all complaint soon ceased. After discharging them from the hospital at the end of about three weeks subsequent to operation, I have had them report to the clinic sufficiently long to assure myself of their positive convalescence. I was sorry not to have been able to operate on a larger proportion of my cases; but as I had to explain to all of the earlier ones solicited that the method I proposed as a cure was in the nature of an original experiment, it was natural that several of them should have refused to submit, in spite of my assurance that I hoped and expected to cure them. Now, however, in consideration of the results accomplished, I do not feel that I have to emphasize the experimental feature of my method, and, consequently, it is probable in the future that a larger percentage of those suffering will choose a speedy relief through operation rather than a tedious one through ex-

pectant treatment. Aside, also, from the speedy cure, the operation insures there is another argument in its favor, and that is the decrease it affords in the danger of grafting on the system cardiac, joint, or other lesions the result of sepsis where through its agency systemic sepsis is promptly eliminated.

The clinical report of my cases is as follows:

Case I.—A young man, twenty-three years of age, whom I found on my service of the City Hospital, when I began my annual visiting there in December last. Five months before, the patient had contracted gonorrhoea, and for the preceding four months he had been so crippled by gonorrhoeal rheumatism that he had been forced to remain in bed, being unable to walk. Both ankles and feet were much swollen and very tender to the touch. Both knees were also similarly involved, and there was sufficient fluid in both these joints to cause a distinct floating of the patellae. The upper extremities were not markedly affected, though sharp pains were frequent in connection with movements of the shoulders and back. Even though the patient kept as quiet as possible in bed, his feet and legs caused him much suffering. As the result of a careful genito-urinary examination, I found that the only existing lesion was confined to the seminal vesicles. Both these organs were markedly indurated and tumefied. In fact the tumefaction was so extensive that it had invaded to a considerable extent the peripheral tissues. The patient was very anxious to get well, and, as he was thoroughly discouraged with the lack of progress he had been making, readily agreed to operation. I accordingly performed my usual operation of seminal vesiculotomy. By the retraction of the rectum, both seminal vesicles were exposed, and longitudinal incisions were made into each, opening their cavities thoroughly. After gentle curettage, both cavities were packed with sterile gauze, and drainage established in the usual manner. The day following the operation the patient reported himself as very comfortable, and free from pain even when he moved his knees and ankles. On my first visit to him, forty-eight hours after operation, I found I could manipulate his knees and ankles without causing him any pain. The fluid in his knees had disappeared and the swelling in his feet was much reduced. At the end of five days the swelling had disappeared from his feet, and he could then make all extreme movements possible while lying in bed, without causing any pain or even discomfort. At the end of a week the drainage from his seminal vesicles was removed, and the tract of the wound was allowed to close. At the end of two weeks I ordered him out of bed. Careful rectal examination then showed a rapid resolution to be taking place in connection with his seminal vesicles. About a week later he left the hospital. After leaving the hospital he reported once a week to me at my Post-Graduate

Clinic for about a month. He was then in robust health, with the appetite of one convalescent from typhoid. On his last visit, I told him he need not report again if he continued well. I have not heard from him since.

Case III.—Man, twenty-six years old, City Hospital service, February, 1905. This patient on entering the hospital had gonorrhoeal rheumatism in both feet and ankles, the parts being markedly swollen and very tender to the touch. The pain on attempting to walk was so great that rest in bed was imperative. About six months previously he had contracted gonorrhoea, apparently his second attack. Three months previously the rheumatic complication in the feet and ankles showed itself, and for a month it was not especially severe. For the last two months, however, it had been in its present state of intenseness, forcing the patient to take to his bed. Six weeks previously he had entered the hospital, but at that time, not being prepared to entertain the idea of operation as a cure for his trouble, he had left the institution. A subsequent six weeks of suffering had, however, forced him to change his mind, and now he re-entered the hospital ready to accept operation. His genito-urinary lesion was a seminal vesiculitis involving especially the lower halves of both organs with a dense circumscribed sclerosis. I performed seminal vesiculotomy, cutting freely through the entire length of the surrounding sclerous tissue, and fully exposing the sac cavities. At the end of twenty-four hours the pain and tenderness to touch had disappeared from the affected parts, and forcible manipulations of the joints could be performed without discomfort to the patient. At the termination of two weeks, the patient left his bed and could walk without difficulty or discomfort. I practiced him walking on his toes and raising himself from a squatting position, keeping his heels off the floor, and found he could perform these exercises without trouble or inconvenience. Rectal examination showed that a resolution had taken place in connection with the genital lesion.

Up to date, February 1, 1908, I have performed the operation of seminal vesiculotomy 101 times without a death. Out of this number the operation has been undertaken 23 times for the relief of gonorrhoeal rheumatism.

In all the 23 cases the operation has had a profoundly beneficial effect on the rheumatism. All have left the hospital in a well or greatly improved condition. In all these cases there was, of course, a seminal vesiculitis to account for the focus from which the systemic absorption originated. Out of the 23 cases 17 were cured as a direct result of the operation and were well when last seen. Of the remaining 6, two got well after the operation and remained so till about a year, when each of them contracted gonorrhoea afresh. Rheumatic symptoms then reappeared. Both of these cases were then seen but once with moderate rheumatism, after which all trace of them was lost. Two more cases after leaving the hospital in a very satisfactory condition immediately resorted to sexual intercourse. The sexual

exercise during the convalescent stage from the operation naturally reinflamed the seminal vesicles and there was a relapse of the rheumatism. In both these cases, however, the rheumatism again disappeared after the effects of the premature sexual exercise had passed off and these cases are now well. Two very chronic cases relapsed, one after being well 8 months after operation and one after being relieved 3 months. In both these cases there was a recrudescent of the tenderness and inflammation in the seminal vesicles. They were destitute individuals and probably exposure and poor living were to blame for the relapse.

In the acute bedridden cases the effect of the operation is most marked. In 24 to 36 hours the pain in the joints disappears, and by the 4th day the swelling. Some stiffness and some atrophy of the muscles is, of course, left after the rheumatic process has disappeared in these acute cases requiring massage and passive movement.

In the chronic cases pain disappears in a week or 10 days, and at the end of two weeks massage can be prescribed. In the very chronic cases it may take two to three months of massage and exercise to limber up the joints which have been robbed of their function. In fact, one has the same problem in these cases which the surgeon has after resolution from a fracture of standing. It is well known that in chronic gonorrhoeal rheumatism massage and manipulation make matters worse whereas in these cases convalescent after seminal vesiculotomy such treatment is beneficial because there is no longer the systemic absorption of the toxine.

In many of the chronic cases the preceding gonorrhoea was so remote an affair that the existing rheumatism had supposedly had no connection with it. In these cases the diagnoses of uric acid, chronic rheumatism allied to the so-called inflammatory form, arthritis deformans, gout, neuritis and neuralgia had been made to account for existing conditions.

The physician or orthopaedic surgeon rather than the genito-urinary surgeon had been consulted by most of these sufferers.

One point I have noticed after operation in quite a number of these cases and that is that in the third week after operation there may be a mild and temporary recrudescence of the rheumatic symptoms whereas in the two weeks immediately after operation there had been a complete recessation of all such symptoms. Such recrudescence I take it is due to a somewhat premature closure of the tract of the incision before a complete elimination by drainage of all the toxines has taken place. In all such cases I have been able to demonstrate by palpation with the finger tip introduced at the rectum considerable remaining tenderness and tumefaction in the region of one or both seminal vesicles. By further rest together with tonics and attention to nutrition such remaining tenderness and tumefaction will spontaneously disappear in most instances provided the seminal vesiculotomy has

been thoroughly performed. In case there should not be a spontaneous disappearance it may be necessary to open the tract of the wound and reintroduce the drainage tubes, keeping these same reintroduced tubes in place till all question of their necessity has disappeared. I have never found it necessary to reintroduce the drainage tubes in any of my cases of gonorrhoeal rheumatism, although on two or three occasions I have had to do so in connection with my seminal vesiculotomies undertaken for the relief of other ailments.

During my December, 1907, and January, 1908, service at the City Hospital I have successfully performed seminal vesiculotomy 8 times for the cure of gonorrhoeal rheumatism. For the collection of this large amount of material in so short a space of time I am under much obligation to Dr. Mitchell, who was my house surgeon for that period. A brief detail of the histories of these cases is as follows:

Case I., H. L. 22, December, 1907.—Helpless and bedridden owing to gonorrhoeal rheumatism involving left leg, hip and thigh. The symptoms were acute and severe. He had been confined to bed 4 months following a gonorrhoea which he had contracted 5 months before. There was a marked seminal vesiculitis, but no other genito-urinary lesion. Seminal vesiculotomy. In this case there was some bleeding 24 hours after the operation for which the incision was partially reopened and repacked. Following this reopening there was some urinary leakage through the wound. This case now 7 weeks after the operation is entirely well of all trace of rheumatism. There is a slight stiffened state of the hip, but this is rapidly disappearing as a result of massage and of walking about. The resolution in this case was somewhat tardy, probably due to the repacking of the wound and the temporary urinary leakage. Patient is now ready to be discharged.

Case II., P. L., 23, December, 1907.—Very acute febrile case of gonorrhoeal rheumatism involving both wrists, left hip and right knee. These joints very much swollen and very tender. Patient in bed and unable to move. The patient had been suffering from this acute attack about one month. This was his second attack. His first one, which had been very tedious, had been 3 years ago, following gonorrhoea. Previous to the present attack he had had a urethral discharge, but whether this was a reinfection or relapsing in character was not clear. Examination showed the present lesion to be a seminal vesiculitis. Seminal vesiculotomy was performed followed by a very quick subsidence of all his rheumatic symptoms. In 3 days all active evidence of the rheumatism had disappeared. Four weeks after the operation the patient left the hospital to sail for Europe. He was completely well, except for a slight stiffness of the right wrist. This was rapidly disappearing as the result of active use of the part.

Case III., V. A., 45, December, 1907.—This patient entered the hospital owing to trouble in connection with his left knee, which he had had apparently for four months. The knee was the seat of a hard inflammatory tumefaction consisting of an exudation. This exudate had caused complete anchylosis. The joint was tender to deep pressure and much pain and tenderness resulted when the weight of the body was put on the joint. The patient gave a past history of several gonorrhoeas or relapsing urethral discharges. His sexual function was also impaired. At the time he entered the hospital he felt that he had no gonorrhoea or urinary inflammation. Medical and orthopaedic forms of treatment had been tried with no beneficial effect and the patient was transferred to my service. Examination showed that he had a chronic seminal vesiculitis, especially in connection with the right sac, the lower portion of which was enveloped in a sclerous infiltration. Seminal vesiculotomy was performed, the sclerous infiltration cut through and the sacs freed from adhesions and freely incised. Painful sensations quickly left the joint after the operation followed more slowly and gradually by a resolution of the hard exudate. With the disappearance of the exudate the joint motion has gradually returned. Now the patient walks about without pain and with much movement in the joint, and this movement is increasing rapidly.

Case IV., J. S. 30.—Operated on early in January. Chronic gonorrhoeal rheumatism of 8 years' standing. Both knees, hips, spine and neck were involved. Back was bent, no mobility to spine and but very little motion to neck. Had been on crutches for a year before operation. Got his first gonorrhoea 9 years ago and a second attack 4 years ago. Both seminal vesicles were thickened and embedded in a peri-seminal vesicular sclerosis. Seminal vesiculotomy was performed five weeks ago. The thickened sclerosed tissue surrounding the seminal vesicles was cut through on each side and the seminal vesicles were freed from surrounding adhesions, after which both sacs were freely incised, packed with gauze and drainage tubes adjusted. In this case owing to the chronicity of the lesion there was no active intra-seminal vesicular catarrh, the sac walls being in fact compressed and somewhat atrophic. A few intra-cellular diplococci were found in a smear, taken from the region of the sac cavity.

One week after operation patient could move his back and neck quite freely without pain. He is now walking about easily and quickly without crutch or cane. The affected joints are still somewhat stiff and the muscles have not yet recovered wholly from their atony, but those symptoms are rapidly disappearing as the result of massage and exercise.

(Cases III. and IV. I exhibited at the Surgical Section of the Academy of Medicine, February 7, 1908.)

Case V., M. T. 29, December, 1907.—This case presented an instance of acute gonorrhoeal rheumatism of 6 weeks' standing. Patient was confined to bed. Both knees and thighs, right wrist and back were involved. The knees and wrist were very much swollen. Patient was thoroughly helpless and in great pain. He gave a history of gonorrhoea 11 years ago, 3 years ago and 3 months ago. The seminal vesicles were markedly sclerosed and evidently recently reinfected. This was his first attack of rheumatism. Seminal vesiculotomy was performed. Three days after the operation patient was wholly free from pain and by the end of 4 days all swelling had disappeared. He could then move his back and legs and wrist without discomfort. In this case at the end of 2 weeks after the operation there was a slight recrudescence of the pain in the joints due to the premature closing of the outside wound, thus checking all drainage. Before the 4th week, however, this recrudence had disappeared, leaving the patient in good order and free from his rheumatism.

Case VI., J. L. 23, January, 1908.—Patient presented a very acute synovitis of the right knee. The joint was greatly distended and there was marked surface redness and tenderness. No movement of the joint was possible. This condition had persisted for 6 weeks and had been uninfluenced by rest or anti-rheumatic treatment. Had had gonorrhoea 5 months before affection in joint appeared. The gonorrhoea had apparently gotten well in 3 weeks and so had not been connected in a causative way with the following joint involvement. There was no urethral discharge and the urine was clear. Rectal examination, however, showed seminal vesiculitis, the inflammatory involvement being chiefly in connection with the right seminal vesicle. I did not at first wish to operate in this case, being fearful owing to the rather slight extent of the seminal vesiculitis, lest the operation would fail to relieve the joint. The patient having seen the benefits of the operation in the preceding cases, begged for the operation and I accordingly performed seminal vesiculotomy. In 3 days all pain, swelling and inflammatory evidence had disappeared from the knee. In fact, on inspection the right knee looked exactly as the left. In this case also at the end of 2 weeks there was a slight and temporary recrudescence of the rheumatism, the right wrist becoming a little stiff. This, however, shortly disappeared, leaving the patient well.

Case VII., J. S. 31, January, 1908.—This case was to me one of the most interesting of the series. For 3 months the man had been unable to move his legs or thighs. He lay perfectly helpless on his back. He had first felt his rheumatism one year before and from that time it had been gradually getting worse. Two years ago he had had gonorrhoea, but stated that he had gotten wholly over the disease in 2 months and had never after

had occasion to think of it. He had taken all sorts of anti-rheumatic remedies and had been treated by orthopaedic surgeons, all to no avail. Rectal examination showed a marked old seminal vesiculitis with much peri-seminal vesicular inflammatory involvement. Seminal vesiculotomy was performed and in 3 days he could move his legs and thighs freely. At the end of 3 weeks on being let out of bed he ran about the ward briskly with no discomfort. This was remarkable as in most such cases the joints are left stiff for a time after the rheumatism has disappeared necessitating massage.

Case VIII., V. L. 27, January, 1908.—This patient had gonorrhoeal rheumatism in both ankles and knees. He was not bedridden, but walked with great difficulty with a stick. Both ankles were somewhat swollen. He had been so affected one month. Two months before he had contracted gonorrhoea. This was apparently his third attack of gonorrhoea. The urethral discharge still persisted at this time. A chronic seminal vesiculitis existed which showed evidence of recent reinfection. Seminal vesiculotomy was performed, followed by a quick disappearance of the rheumatic symptoms. This case at the time of this report is ready to leave his bed.

Before performing seminal vesiculotomy on any of these cases, I have endeavored to exclude tuberculosis as a complicating element through the employment of the Calmette and tuberculin tests. Have these tests in any instance indicated tuberculosis I have refrained from operation.

From the results obtained in the comparatively large number of cases operated upon I now feel that the efficacy of this form of treatment is established.

Preceding articles, bearing on seminal vesiculotomy, by the same author, are as follows:

May, 1901, "Journal of the American Medical Association."—"A New Operative Method to Expose the Seminal Vesicles and Prostate for Purposes of Extirpation and Drainage." A Preliminary Report.

May 21, 1904, "Medical Record," New York.—"Operative Surgery Applied to the Seminal Vesicles. A Demonstration of Some New Principles."

October, 1904, "The Post-Graduate."—"Seminal Vesiculotomy. The Author's Operation."

June, 1905, "Annals of Surgery."—"The Relation of Gonorrhoeal Rheumatism to Seminal Vesiculitis and Its Cure by Seminal Vesiculotomy."

Vol. X., No. 3, "American Journal of Dermatology and G. U. Surgery," St. Louis.—"A Further Report on the Cure of Gonorrhoeal Rheumatism Through Seminal Vesiculotomy."

Vol. XI., No. 9, "American Journal of Dermatology and G. U. Surgery," St. Louis.—"A Plea for the Sexual Protection of Young Boys."

December, 1906, "The American Journal of Urology."—"Operative Cure for a Hitherto Unrelieved Class of Cystitis."

AN AID TO THE DIAGNOSIS OF KIDNEY ABSCESS.

By Robert Holmes Greene, M. D.,

Attending Surgeon, Genito-Urinary Division.

Through the inventive genius of some members of my house staff at the City Hospital, in the spring of 1906, my attention was called to a method which is of aid in the diagnosis of kidney abscess.

They had made the diagnosis in the suspected case, that of a middle aged man, with pus in his urine and some evidence of sepsis, that an abscess was located in one of his two kidneys, by observing that when pressure was made on one side, over the course of the ureter, the amount of pus in the urine materially increased, while massage carried along over the course of the ureter on the other side failed to increase the amount of pus there present.

I operated on the evidence furnished by this massage, on the suspected side, and found a large abscess at the back of the kidney. In looking up the literature of the subject, I was unable to find at the time, any article in the English language upon it.

Two Italians, Giordano, of Venice, and G. Nicholich (La Semaine Med., March 30, 1904), had discussed this procedure and had made useful observations concerning it. It has since then been mentioned in a text-book by the author, and the method somewhat elaborated by him. (Diseases of the Genito-Urinary Organs and the Kidneys, by Greene and Brooks, W. B. Saunders Co., Philadelphia, Publishers, 1907.)

The procedure is as follows:

First (Figs. 1 and 2): Stroking and pressing with one hand from the region of the kidney over the course of the ureter down to the bladder. Second (Fig. 3): Vibratorial massage over the same region, one hand resting on top of the other. These two procedures are well illustrated in the accompanying cuts made expressly for this report.

A rough diagnosis can be made by performing this massage, on one side in a suspected case, and observing if pus is found, or amount of pus already present in the urine is increased by such massage.

A little more elaborate method is as follows: Patient's bladder being clean and empty, massage over the kidney, and along the course of the ureter, and then collect and examine the urine, then bladder being again washed out, massage on the other side, collect the urine, and compare the two specimens, or a catheter may be left in the previously washed out bladder, and the effect of the massage from either side observed. This massage, if it causes bloody urine, may also be an aid in the diagnosis of kidney calculi.

FIG. 1.

METHOD OF EXPRESSING PUS FROM KIDNEY PELVIS INTO THE BLADDER.

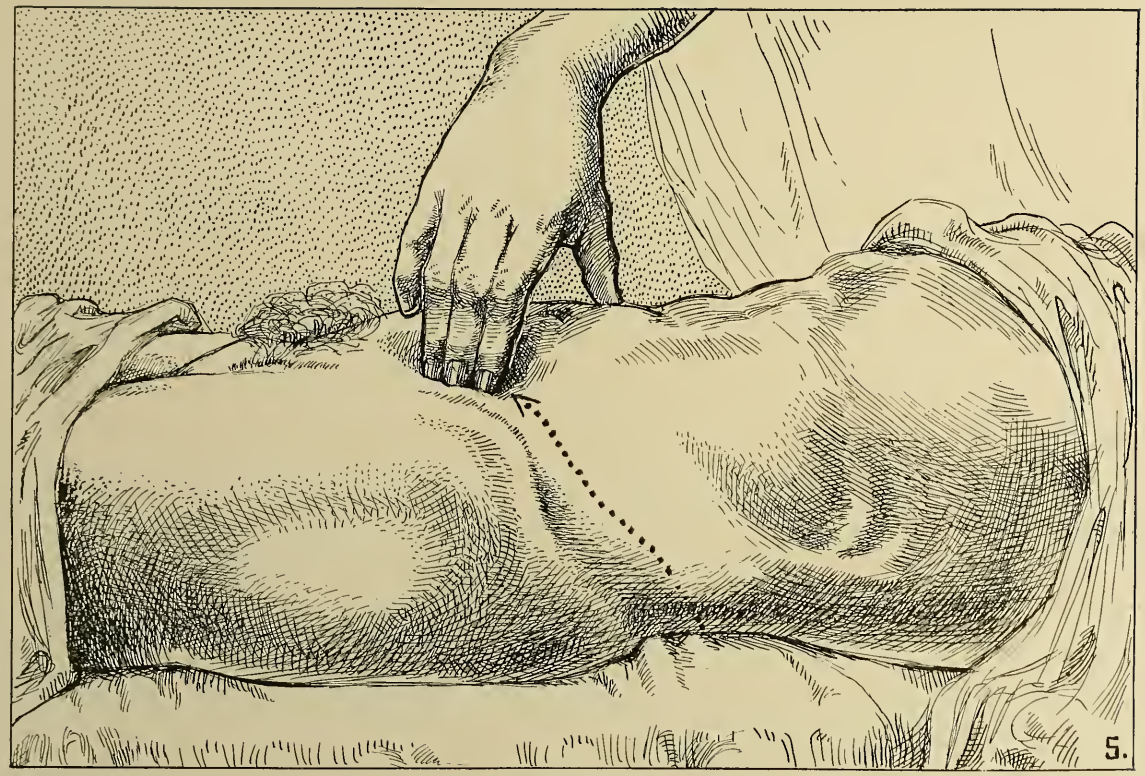


FIG 2.

METHOD OF EXPRESSING PUS FROM THE KIDNEY PELVIS INTO THE BLADDER, CONTINUATION OF FIG. 1.

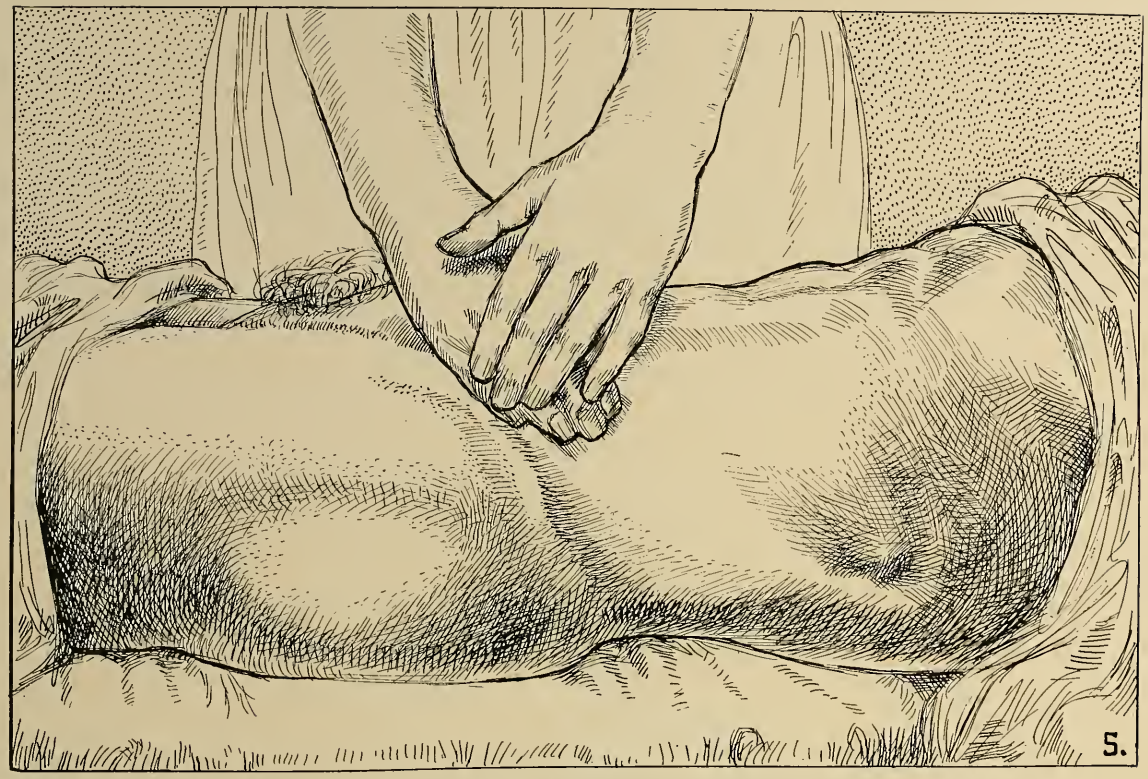


Fig. 3.

VIBRATORY METHOD OF EXPRESSING OR MASSAGING PUS FROM THE RENAL PELVIS INTO THE BLADDER.

V.—Neurological Division

ALTERNATE HEMIPLEGIA—GUBLER-WEBER TYPE.

By Smith Ely Jelliffe, M. D.,

Attending Neurologist.

Unusual types of hemiplegia are of sufficient interest to report, if only in brief. Even when not coming to autopsy, such reports may be of service, both as affording opportunities for anatomo-clinical differentiation, and as offering an occasion for the recording of features which, while not new, are sufficiently unusual to claim attention.

It is for this reason, and as giving a record for future reference, should one case here noted come to autopsy at some future date, that the following histories of cases exhibiting the Gubler-Weber syndrome are given.

But few communications on this syndrome have appeared in English, since the study of Weber himself in 1863 (Med. Chir. Transactions, 1863) (American literature seems to be barren of even case reports), and it is usually held that this author was the first to discuss this particular form of crossed hemiplegia; but such is not the case. Gubler, in his "Memoir sur les paralysies alternes en generale et particulierement sur l'hemiplegie alterne avec lesion de la protuberance annulaire," which was published in 1859 (Gazette hebdomadaire), following three years after his first contribution to the more general subject of paralysis, due to lesions below the crossing of the seventh nerve (Gubler-Millard syndrome), calls attention to the fact that Luton had previously described the condition of a paralysis of the oculomotor of the left side with a total right hemiplegia, and had localized the lesion in the left cerebral peduncle. This, perhaps the earliest case recorded, is worthy of reproduction, since Weber had entirely overlooked Gubler's study, and few writers since have called attention to it, save d'Astros. (Revue de med. 14, 1894.) The earlier text-book recognition by Gendrin and the monograph by Koechlin should not be overlooked.

History of Luton's Case—Auguste D., musician, entered the Salpétriere, April, 1858. Service M. Briquet.

A first attack of paralysis had occurred six months previously, involving the left side of the body. This paralysis disappeared almost entirely, and the patient was able to take up his occupation. On arising on the morning of the 10th of April, 1858, he was suddenly taken with a second attack. This came on without loss of consciousness, there being a general, but not complete paralysis on the left side of the body. Motility is chiefly involved, the sensibility being somewhat reduced when compared with the opposite side. There was slight deviation of the lips; the protruded tongue is directed to the left in an evident manner, but may be moved in all di-

rections. The left nostril is slightly dilated. The left eye is wide open, and cannot be closed. The pupil is mobile, vision clear. The right eye is completely closed and cannot be opened; the right pupil is widely dilated, immobile. The eyeball cannot be raised up nor down, no inward movement, but it moves freely outward. The alternating movements of rotation, following the antero-posterior axis are doubtful. The vision in this side is obscure. He sees objects vaguely, but without precision. When the patient looks with both eyes, he sees double.

The condition remained stationary for some time. Toward the end of September the patient presented an access of temperature, which was intermittent, following catheterization, and died in coma, five months after his admission.

Autopsy—This showed an atrophy of the right third nerve, at its egress in the interpeduncular space, it being diminished in diameter to about one-half of that of the oculomotor of the other side. The right peduncle, seen from the outside, seemed flattened, patulous, and less rounded than that of the opposite side. Its softening and fluctuation was in contrast with the firm consistence of that of the opposite side. Sections of the right peduncle and neighboring regions showed that the entire substance was reduced to a non-homogenous boil of a grayish whiteness at certain points, and of a cafe au lait color in others, and of a blackish tint in the part corresponding to the locus niger; that this focus of softening is badly limited, that it occupies the depth and width of the peduncle, and extends upward to the neighboring region of the optic thalamus.

In the right cerebral hemisphere two other areas of softening were present; one at the extremity of the sphenoidal lobe, the other at the summit of the occipital lobe. These areas have the size of a nutlett. They were well walled off, and their limits were not diffuse. There was about 60 grams of clear limpid liquid in the ventricle. There was a slight spreading of blood into the lateral ventricles and the arteries at the base were atheromatous.

There were chronic inflammations in the urinary organs, small abcesses in the two kidneys; two areas of gangrene of the right lung, and a gangrenous and purulent area in the right lung. A passive dilatation of the heart. Luton ascribed death to the secondary infection.

Since this case of Luton's there have been a number of others reported, an extended analysis of which has been given by d'Astros (Rev. de Med. 14, 1894).

Believing that the Gubler-Weber syndrome is seen rarely, the two case histories to be reported seem worthy of record, as they present certain features concerning which little is known.

The first case that I wish to report is that of Albert Drecker, a laborer, 44 years of age, single. He was admitted to the City Hospital April 19,

1908. On his admittance the patient was in deep coma, irresponsive, and had come from Bellevue Hospital in that condition, evidently suffering from an apoplectic stroke as his left side was completely inert. He lay in this condition for about three weeks, being unapproachable, comatose and almost dying; pulse 60, respiration 16 for much of the time, at times the pulse was as low as 50.

Even in this condition, there were marked evidences of a complete left-sided hemiplegia, with ptosis of the right eyelid and dilated right pupil, and external squint of the right eye.

In the first week in May the patient came partially out of his coma for four or five days, was unintelligible, very confabulatory and fabricating, rambling and incoherent. He then seemed to have another access of bleeding, and went into deep coma, with rapid, irregular and feeble heart action, from which, however, by the 20th of May, he had made a partial recovery.

From this time on his mental state began to clear, although at the present time of writing, May 27, 1908, it is not sufficiently clear to warrant the acceptation of all his statements.

He is single, denies luetic infection, but admits to being a heavy drinker. He remembers almost nothing concerning the incidents antecedent to his attack, and very little trustworthy history is obtainable.

His physical examination at the present time shows:

Cranial Nerves.

First Nerve—There is no impairment of smell, he recognizes aromatic odors.

Second Nerve—Sight is somewhat impaired on the right side, unimpaired on the left. There is no diplopia, and no hemianopsia. The blurring on the right side is largely due to paralysis of accommodation.

Third Nerve—This shows marked involvement on the right side; right ptosis. The only fixed position of the right eye is upward and outward, and the movements are limited, the external rectus and the superior oblique apparently being the only muscles spared. Marked dilatation of the right pupil, with diminution of reaction to light on the right side is present. The ocular movements of the left side are normal. The left pupil reacts to light and accommodation, and shows some consensual light reflex; Wernicke's hemiopic pupillary phenomenon not elicitable.

Fourth Nerve—Apparently uninvolved on either side.

Fifth Nerve—No sensory disturbance in the fifth nerve distribution on either side.

Sixth Nerve—No anomalies in the sixth nerve. Patient's mental state prevents accurate tests for diplopia.

Seventh Nerve—There are slight traces of a facial palsy on the left side. For the first three weeks, during which the patient was unconscious, he kept his left eye open most of the time, and winking did not seem to be spontaneous. At the present time, however, there seems to be no obicularis paresis, and the lower branches of the seventh are not markedly involved. The mouth was drawn a little to the right, and there was partial obliteration of the left nasolabial fold, but at the present time there are only slight residua of left facial involvement.

Eighth Nerve—No deafness and no hallucinations.

Ninth Nerve—No involvements.

Tenth Nerve—The heart action is regular; there is no marked disturbance of breathing at the present time. During the second attack the patient had a very weak, feeble and irregular heart action, and the general impression given was that of cardiac disturbance.

Eleventh Nerve—No involvement.

Twelfth Nerve—Tongue—left side is a little softer than the right. Fibrillary tremors are marked. The tongue protrudes very slightly to the left, but is easily movable in all directions.

Spinal Nerves—Marked paralysis of the left arm; it is flaccid, swollen and oedematous, and there is a complete loss of all reflexes. No marked sensory disturbances are obtained, although in this respect the patient's mental state does not admit of an exhaustive analysis. The left leg is less markedly involved than the left arm. The patient is able to flex the thigh and leg, but the movements of extension are very weak. There is distinct atrophy and loss of knee jerk of the left side. There is no Babinski. Slight false clonus. The muscles of the right side are not involved. Knee jerk, wrist jerk, elbow jerk all present on the right side. No clonus, no Babinski, no Oppenheim. No sensory anomalies.

This case then presents a clear-cut case of the superior alternate paralysis so well described by both Gubler and Weber. It is now an interesting feature that the patient still shows marked somnolence, and there is absence of any hypertonic phenomena on the paralyzed side, four weeks after the original injury.

Case 2—John Lindsay, age 29, born in the United States, single. The patient was admitted into ward five of the City Hospital on December 7, 1906, first entering Dr. Quimby's ward, and later that of Dr. Collins, through the courtesy of both of whom I was permitted to see the case in the first instance.

The patient was a laborer, markedly alcoholic. He had suffered from gonorrhea several times, and had had syphilis three years previously.

In the immediate history, the patient complains of recent headaches. There was absence of rheumatism, malaria and typhoid. In childhood he suffered from measles, chicken pox, whooping cough and scarlet fever. Has not heard well in the right ear since he had scarlet fever. The patient, however, gave a history of having fallen a distance of twelve feet six months prior to the onset of his present illness. A week or ten days before his admission to Bellevue Hispital he fell a distance of about three feet, landing on his left shoulder. Shortly following this, although not immediately, he was taken to Hudson Street Hospital suffering from a left-sided paralysis. He remained in the Hudson Street Hospital for five weeks, and was then transferred to Bellevue, where he remained for six weeks, the fragmentary note on transfer being that he had a partial paralysis of the left arm and leg and ptosis of the right eye.

The patient is a well nourished and well built man. At the time of making these notes (May 19, 1906), he is about the ward, is feeling well, but suffers from a left-sided paralysis, and from external strabismus of the right eye, with partial ptosis.

Physical examination shows the lungs to be unaffected; the heart is not enlarged; its action is slow, heavy and lagging; the systolic beat at the apex and the second and pulmonic sounds are accentuated; the aortic sound is sharp in character. The pluse is full, moderate, compressible, both sides synchronous. Abdomen negative.

Cranial Nerves.

First—No disturbance of smell.

Second—No disturbance of sight, no diplopia, no hemaniopsia. Patient had had diplopia earlier, and now shows it at times on testing.

Third—There is a distinct ptosis of the right eyelid. The right pupil does not react to light or accommodation, and is larger than the left. The left pupil reacts freely to light, and to accommodation. The patient does not see well with the right eye, as there is partial paralysis of accommodation. Note by internes later shows some reaction of right pupil to light and accommodation. The position of the right eyeball is outward and downward; movements in other directions are impossible.

Fourth Nerve—No palsies observed.

Fifth Nerve—No changes in sensation.

Sixth Nerve—Superior oblique action apparently present.

Seventh Nerve—The patient had a marked facial palsy on the left side when admitted. This improved considerably, but at the present time there is a distinct loss of the naso-labial folds on the affected side. The mouth is

drawn to the right (to the left in hospital history). There is a distinct loss of movement on the left side of the face.

Eighth Nerve—Impairment of hearing on the right side, held to be the effect of scarlet fever.

Ninth Nerve—No known involvement.

Tenth Nerve—No involvement.

Eleventh Nerve—Some weakness in the muscles supplied by the eleventh nerve.

Twelfth Nerve—The tongue protrudes to the left. Left side is markedly atrophied. Speech is thick, but comprehensible.

There is marked paralysis of the entire left side of the body attended with atrophy, increased reflexes throughout, with increased ankle jerk, knee jerk, clonus and Babinski on the left side. The leg is freely movable, the arm less so and is oedematous. The right side shows some tenderness of the thigh muscles, with sluggish reflexes. No oedema of either lower extremity. No sensory disturbances either to heat, cold or pain. The fundus is negative. Mental state is stupid. Patient sleeps a great deal in the wards. He left shortly after my examination, went home, and died some six months later; cause unknown.

Case 3—Annie Zimmerman, aged 30, born in Austria, was admitted to the City Hospital on the 4th of June, 1906, and entered the Neurological Ward on the 1st of October, 1906, under the service of Dr. Pritchard, to whom I am indebted for the privilege of reporting the case.

The patient was in a semi-stuporous condition when first seen on the 1st of October. On October 3 this condition became very marked and the patient could not be roused. This gradually cleared up, but she still remained very drowsy, and seemed to sleep practically all the time when not disturbed. She was unable to give much of a history, although when she came into the hospital she said that for the past three years she had been suffering from dizziness and had had attacks when she would fall to the ground. For a year or so she had been unable to do much work, and complained of her feet and hands. The patient was of limited intelligence.

There was no history obtainable of an acute onset.

Examination showed a poorly nourished woman, apparently 35 years of age, skin pigmented. Sounds in the chest were practically normal, there being only a slight dullness over the left apex. There heart sounds were clear. There were no murmurs; pulse was regular and of good volume, and tension not exaggerated.

Examination of the nervous system showed the following:

First Nerve—No anomalies of smell.

Second Nerve—The patient did not see very well in the right eye, owing to paralysis of the accommodation.

Third Nerve—Ptosis of the right eyelid, slight lateral nystagmus, right pupil larger than the left, does not react to light or accommodation. Restriction of pupils of the right eyeball impossible to move it inward. It occupies an exteral upward position.

Fourth Nerve—The patient not in a condition to respond to tests for diplopia.

Fifth Nerve—No changes observed.

Sixth Nerve—

Seventh Nerve—Marked facial palsy on the left side, involving also branches of the facial. Jaw pulled somewhat to the right. Tongue protruded to the right, speech thick, somewhat incoherent. Patient unable to repeat complicated phrases.

Eighth Nerve—The patient could hear watch at five inches on the right side, on the left could not hear watch at all.

Ninth Nerve—

Tenth Nerve—No anomalies of respiration or heart action.

Eleventh Nerve—

Twelfth Nerve—Taste apparently unaffected.

Spinal Nerves—There is marked hemiplegia of the entire left side. The left arm is weak in all its movements, tendon reflexes are exaggerated on the left side. There are no changes in the tactile or temperature sense. Slight ataxia of the hands and arm on left side. Right upper extremity is normal. The lower extremities show paralysis of the left side, with increased tendon reflexes, with ankle clonus and slight Babinski on the left side. No anesthesia to touch or temperature. Both sides, deep sensibilities normal.

There is slight rigidity throughout the whole left side, although muscular power is not entirely gone. All the movements are slower on the left side. There is slight anesthesia to touch and temperature on the right side of the face, although it is difficult to say just how much of this is stupidity or actual observation.

The patient remained in the hospital for several months, in practically the same condition; on November 30, 1906, got up a pneumonia and died December 10, 1906. Autopsy was refused.

The parallelism in these two cases is very striking. Both are typical illustrations of the Gubler-Weber syndrome, the first case showing the

symptoms at the onset, with flaccid paralysis and loss of knee jerk, no Babinski, no contractures, as yet; whereas the second case shows the later stages when contractures and spasticity have set in, with increased reflexes throughout. It is of interest that both patients were under 50.

Since hemorrhage, or softening of the cerebral peduncles is due in the first place to a rupture, and in the second to an obliteration of the arteries that supply the peduncle, a few words concerning the distribution of these arteries in the different regions of the peduncle may not be out of place. Alezais and d'Astros (Journal de l'Anatomie et de la physiologie, 1892, p. 519), have shown, very distinctly, that the posterior cerebral artery and its branches give rise to practically all the arteries which supply the peduncle. When obliteration takes place, the general softening that results is conditioned very largely by the frequent and full anastomosis of the arterial trunks which, in this situation, are very rich. So far as hemorrhage is concerned, the only thing that need concern one are the nourishing arteries that go directly to the peduncle. These arise, for the most part, from the trunk of the posterior cerebral, usually in the ascending portion before the giving off of the posterior communicating artery; may be in the recurved portion of the circumpeduncular. A certain number of the arteries that go to nourish the peduncle come off from a portion of the posterior cerebral, which coming from the ascending branch, are distributed to the corpora quadragemina and later pass into the peduncle. D'Astros has termed this artery the peduncular jumelle. Several other branches rise as low down as the superior cerebellar, and as high up as the posterior communicating, or even the middle cerebral.

All of these arteries d'Astros divides into five groups.

(1) The internal peduncular, the most numerous, the greater number of which come directly from the posterior cerebral, enter the internal portion of the peduncle, but do not pass beyond the substantia nigra.

(2) Antero-external peduncular arteries, numerous, which generally terminate in the lower portion of the peduncle, rarely traversing the substantia nigra, to enter the upper levels. They may be found coming as far up as the thalamus.

(3) The superficial arteries, distributed to the sulcus.

(4) Superior peduncular arteries, few in number.

(5) Arteries which supply the corpora quadrigemina. These are supplied by a very rich plexus, constituting the termination of the peduncular jumelle artery, occasionally the superior cerebellar, occasionally the terminal branch of the posterior cerebral, each carry a branch of this plexus. All of these arteries are very fine, their rupture is very possible, but experi-

ence shows that it is comparatively rare, and so far as the corpora quadrigemina are concerned, hemorrhages within the substance are very unusual.

Two of the series of branches are of special interest. Those which only traverse the peduncle to supply neighboring regions, the so-called optic arteries, internal and external. These are of less importance in the consideration of our particular subject than the second series, made up of the arteries of the third nerve nucleus.

Duret's studies (Archives de Physiologie, 1873), d'Astros (Societe de Belgique, June 4, 1892), later worked over by d'Astros, seem to show that these arteries which supply the third nerve nucleus, belong to an absolutely independent system from the other peduncular arteries, especially the superior peduncular arteries and are the unique supply of the nuclei of the third and fourth nerves.

As to the question of hemorrhage or softening, it may be said that softening in the cerebral peduncle due to emboli are probably rare in the vertebral system. But, arteritis, especially of syphilitic origin, is known to involve the basilar with great frequency. It has been seen that the chief nourishing arteries for the peduncle are derived from the posterior cerebral, and that the internal peduncular arteries, the arteries of the oculomotor and the internal and posterior optic are chiefly terminal arteries, not supplied with important anastamoses—obliteration of these causes a much wider syndrome than that which we have under discussion.

It is to a hemorrhage of the artery of the third nerve nucleus that we must look for the interpretation of the lesion in both of our cases. The ophthalmoplegia is complete in each, both external and internal mechanisms being involved. The hemiplegia is largely to be interpreted as secondary and as complicating.

Further interest attaches itself to the record of somnolence, and to coma in both cases.

In a note made by Dr. Collins on Lindsay, it is recorded that the patient is very sleepy most of the time. Although this observation is made many months after the injury, it is of interest to recall that in two observations by Rickards and Leube, somnolence, or an intellectual torpor, or even an incoherent delirium, was present in their patients. In my first case, this incoherent delirium was a very marked feature. He had it at the time of making these notes, and his somnolence is also a characteristic feature. In Rickard's case (Brit. Med. Jl., April 4, 1886), the patient was very sleepy, spoke only when questioned, answered in an incoherent manner, did not care to take any nourishment, saying that he was being poisoned. Leube's patient (Deut. Arch. f. klin. Med., 1887), was in a marked condition of somnolence, did not reply to questions, although neither aphasic, nor anarthric. Both of these patients, however, died very shortly after the

apoplexy. My first patient is still sleepy and incoherent, two months after the onset.

D'Astros believes this somnolence to have some relation to the position of the hemorrhage, and locates it on the internal aspects of the peduncle, extending above the substantia nigra in its upper portions, rather than being a hemorrhage of the internal portions of the peduncle in its lower portions. In this latter region partial involvement of the opposite third nerve is likely, causing a conjugate deviation. Further, when the lesion is more superficial the paralysis of the third nerve is more likely to be partial rather than complete.

ON HERPETIC INFLAMMATIONS OF THE GENICULATE GANGLION. A NEW SYNDROME AND ITS COMPLICATIONS.†*

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Under the general heading of herpetic inflammations of the geniculate ganglion of the facial nerve, I have brought together for the first time three separate groups of cases; each group presenting distinct and clearly defined characteristics, but showing various combinations and transition forms. Their union I believe constitutes a new and distinct clinical entity.

The pathology of this affection is identical with that of herpes zoster, of which it forms a part, the distinguishing features of the clinical picture depending entirely upon the ganglion involved and the nature of the structures surrounding it. Heretofore the only recognized seat of an herpetic inflammation on a cranial nerve was that of the Gasserian ganglion of the trifacial. Herpes zoster in the distribution of one or more of its branches was the result. I believe, however, that the geniculate ganglion situated in the depths of the internal auditory canal at the entrance to the Fallopian aqueduct may be the seat of this specific inflammation. The peculiar situation of the ganglion within the confines of a bony canal and its immediate relationship to the facial and the close proximity of the auditory nerve are responsible for the characteristic complex of symptoms which results.

As was long ago pointed out by Bärensprung and is now definitely established by the elaborate clinical and pathological researches of Head and Campbell, the primary or infectious form of herpes zoster is dependent upon

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a specific inflammation of one or more of the posterior spinal ganglia. Head suggested the name, posterior poliomyelitis for the affection, and certain points of resemblance were drawn between it and acute anterior poliomyelitis. The ganglia involved are swollen by the products of inflammation and by extravasation of blood, and in some cases even the sheath and nerve roots may be involved in the inflammatory process. In very rare instances the anterior or motor root, resting upon the sheath of the ganglion may be implicated and paralysis result.

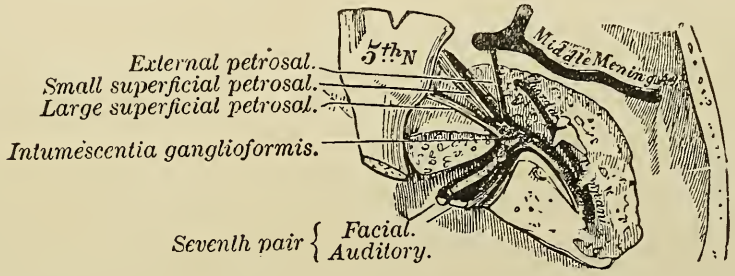

Fig. 1.

The course and connection of the facial nerve in the temporal bone. (Gray's anatomy.)

Such paralytic complications are, however, extremely uncommon, and the total number of recorded cases is so small that they may be given in a few words. Herpetic inflammation of the Gasserian ganglion has given rise to palsies of the neighboring ocular nerves in 15 cases; in 12 cases the oculo-motor was the nerve affected, in two cases the trochlear and in one case the abducens. Paralytic complications of spinal zoster are recorded even less frequently. Arm palsies were observed in eight cases, to which I can add a personal observation, and a localized paralysis of the abdominal muscles in one case. Where the herpetic inflammation attacks the geniculate ganglion, palsies are of much more frequent occurrence than in any other localization of the disease. I have collected 56 cases from the literature to which I can add four personal observations, making a total of 60 cases in which palsies accompanied the inflammation in this situation. This I would attribute to the peculiar location and relations of the ganglion involved.

Clinically the cases of geniculate herpes resolve themselves into three groups. The simplest expression of the disease is a herpes of the auricle and external auditory canal. *Within this skin area is to be found the zoster zone for the geniculate ganglion.* In another group of cases there is added to the aural herpes a paralysis of the facial nerve. This I explain by pressure of the inflamed ganglion or in some cases by a direct extension of the inflammation to the nerve. The most interesting, as well as the most severe,

type of the disease occurs when the acoustic nerve is also involved. In this form there are with herpes auricularis and facial palsy, various auditory symptoms, ranging in severity from tinnitus aurium and diminution of hearing to the more severe forms of acoustic involvement as seen in Ménière's syndrome. In these cases I assume that the inflammatory process has extended to the auditory nerve which is enveloped in the same sheath, and courses in the same canal as the facial nerve.

Each of these groups has separately been the subject of careful study by many observers; but their intimate clinical relationship to one another, their common pathology and their common seat of origin, the geniculate ganglion of the facial nerve, has not heretofore been recognized.

I have already expressed my belief that the geniculate ganglion has its cutaneous representation and zoster zone in the auricle and external auditory canal, and that herpes zoster in this region may have facial and auditory complications. In regard to the distribution of the zoster I wish to lay especial stress upon this fact, that while these neural complications occur in auricular herpes, they also accompany herpes facialis, herpes occipitalis and cervicalis. It will be observed that in these forms of zoster, the herpes facialis and herpes occipito-collaris, the zoster zones lie immediately in front of and behind that which I have indicated as the geniculate zoster zone. Thus the Gasserian, geniculate and 2d and 3d cervical ganglia may be regarded as forming a ganglionic series or chain, their cutaneous zones corresponding to the face, ear, head and neck.

It is not difficult to find an explanation for the occurrence of neural complications (facial and auditory) in those forms of zoster in which the eruption is not in the auricle and therefore is not in the geniculate area. The pathology of herpes zoster is such that while the inflammation predominates or is centered chiefly in one ganglion, other ganglia immediately above and below this central focus may also show inflammatory changes, but in a lesser degree. In fact, a series of ganglia may show milder degrees of inflammation. So that while one ganglion may be regarded as the central focus, evidences of inflammation are not infrequently present in recent cases, in the ganglia immediately above and below, diminishing in intensity from the central lesion.

As the Gasserian, geniculate, and upper cervical ganglia form a continuous system of cutaneous zones, so they may be regarded as forming anatomically a ganglionic series. It is therefore quite natural that if the chief focus of inflammation is centered in the Gasserian, subsidiary inflammatory changes may occur in the other ganglia of this group, the geniculate, and the upper cervical. In such an event the presence of facial and auditory symptoms with herpes occipito-collaris or herpes facialis would be readily explained. As a further evidence of multiple involvement of this

group of ganglia may be cited cases in which the zoster eruption covers two distinct and separate zoster zones.

An idea of the relative frequency of these neural complications and their associated herpetic eruptions may be obtained from the following statistics. In the 60 cases at my disposal for analysis, all had a facial palsy of the peripheral type. In 19 of these cases irritative or paralytic symptoms of acoustic origin were present. In 32 cases the cutaneous manifestation was herpes occipito-collaris, in 12 cases herpes facialis, in 12 cases herpes auricularis, in 3 cases a combined herpes auricularis and occipito-collaris, in 1 case herpes facialis and occipito-collaris combined. I may also add that I have found but one case of herpes zoster with an associated facial palsy in which the eruption was not facial, auricular, or occipito-cervical.

With these introductory remarks I will proceed to a more detailed description of the anatomy, pathology and symptomatology of the affection.

Anatomical Considerations—Before proceeding to the clinical aspect of my subject, it is of great importance that certain facts pertaining to the anatomy of the facial nerve and its ganglion should be known. The facial

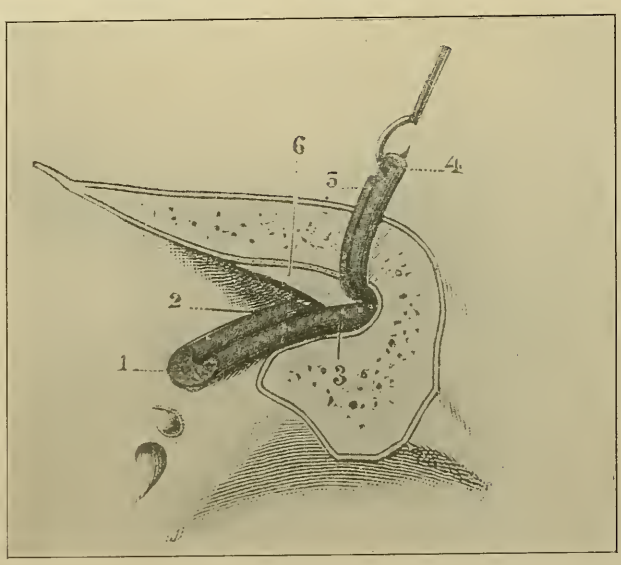

FIG. 2.

TESTUT.—The auditory nerve in the internal auditory canal. 1. Auditory nerve. 2. Cochlear-branch. 3. Vestibular-branch. 4. Facial. 5, Nerve of Wrisberg.

nerve clinically is generally regarded as a motor nerve. This from the anatomical standpoint is not the case. Histological and embryological investigations during the past ten years have shown conclusively that the facial is a mixed nerve, possessing an afferent or sensory portion, which

is the nerve of Wrisberg, and a ganglionic structure, the geniculate, analogous in structure to the spinal ganglia of the posterior roots, the cell type of the geniculate corresponding exactly to that of the spinal and Gasserian ganglia.

The facial enters the internal auditory meatus in company with the acoustic and the nerve of Wrisberg. Within the auditory canal the 7th which is above rests in a slight concavity formed by the acoustic, the *pars intermedia* or nerve of Wrisberg lying between. All these nerve trunks are united and held together by a common sheath of connective tissue. At the bottom of the canal the acoustic divides into its cochlear and vestibular branches, the nerve of Wrisberg and the facial entering the aqueduct of Fallopius. Immediately after its entrance the facial swells into the *intumescentia ganglioformis,* in which the nerve of Wrisberg takes its origin. From this point the facial nerve proper is continued to its peripheral distribution

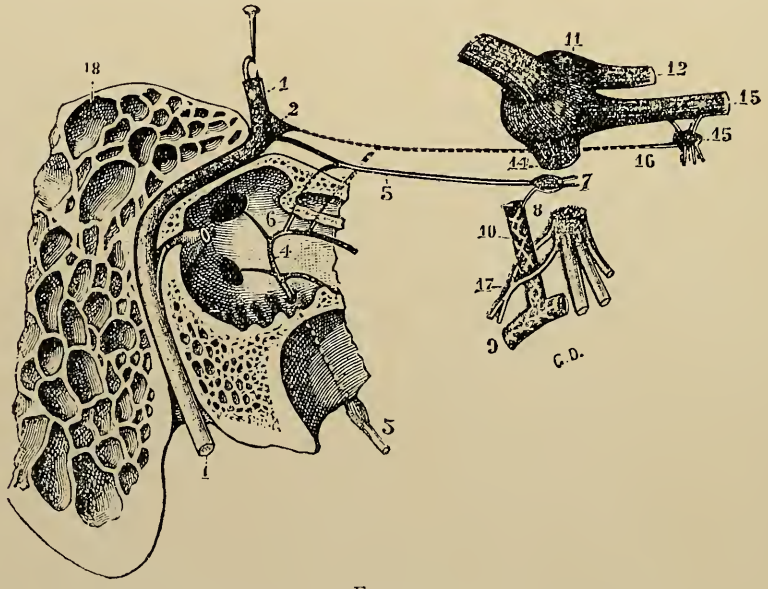

Fig. 3.

TESTUT'S ANATOMY.—The facial nerve; geniculate ganglion and relations with the otic. 1. Facia. nerve. 2. Geniculate. 3. Glossopharyngeal. 4. Jacobson's nerve. 5. Small superficial petrosal 6. Small deep petrosal. 7. Otic ganglion. 8. Sympathetic ramus. 10. Middle meningeal artery 11. Gasserian ganglion. 12. Ophthalmic branch. 13. Superior maxillary. 14. Inferior maxillary. 15. Spheno-palatine ganglion. 16. Vidian nerve. 17. Auriculo-temporal nerve.

through the Fallopian aqueduct. As it lies in the entrance of the Fallopian aqueduct and the *Hiatus Fallopii,* the geniculate has important connections with other ganglia. It is connected with the spheno-palatine ganglion (Meckel's) through the great superficial petrosal nerve, and with the otic

ganglion (Arnold's) through the small superficial petrosal nerve. At their entrance into the medulla oblongata the 7th, 8th and *pars intermedia* have the following relations; the *pars intermedia* lies between the facial and the internal root of the acoustic. It sometimes joins one, sometimes the other of these two nerves as it enters the bulb, terminating in the *fasciculus solitarius* and the central gray column of the medulla. (Fig. 3.)

From this brief resume of recognized and well founded facts it will be observed that my views regarding the affection under discussion finds only encouragement and support.

As the specific infection of herpes attacks only cells of the spinal ganglion type, the geniculate may very properly be brought within the sphere of its influence. Furthermore the intimate relations existing between the facial, the geniculate ganglion, and the terminal division of the acoustic

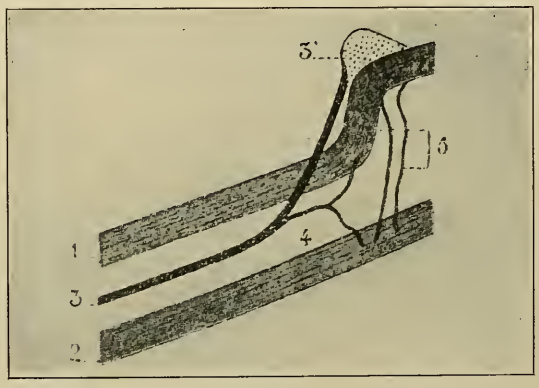

Fig. 4.

TESTUT.- Anastomoses of the facial and auditory nerves. 1. Facial. 2. Auditory. 3. Nerve of Wrisberg. 3^1. Geniculate ganglion . 4. Internal anastomoses. 5. External anastomoses.

would render all these structures liable to involvement when the seat of an inflammatory process; all the more because they are lodged in the depths of an osseous canal, within a common sheath, which would tend to resist expansion and increase the effect of pressure. (See Fig. 4.)

It may be added that the geniculate ganglion varies in size. In some subjects it is scarcely visible to the naked eye; in others the swelling is double the calibre of the nerve. This may well have a certain influence in determining the severity of the case, anatomical peculiarities of the canal contributing.

The Zoster Zone for the Geniculate Ganglion—Admitting the sensory nature of this ganglion, its analogy to a spinal ganglion and its probable involvement in cases of herpes zoster, it is still necessary to demonstrate the

existence of a cutaneous area on the head or face to represent it. This area must be independent of other recognized zoster zones and in it should be found the zoster zone of the geniculate. This zone, I believe, is situated within the auricle and external auditory canal. My argument bearing on this part of the subject is briefly as follows: The peripheral innervation of the external ear is effected through the fifth nerve, branches of the cervical plexus, and the auricular branch of the vagus. The anterior half of the auricle and the superior and anterior walls of the external auditory canal are innervated by the auriculo-temporal branch of the trigeminus nerve. This nerve is a branch of the inferior division of the trigeminus, and I wish particularly to emphasize the neural connections existing between it and the geniculate ganglion through the otic ganglion and the small superficial petrosal nerve. The otic ganglion rests upon the inferior maxillary division of the fifth, just below its origin from the Gasserian. The posterior surface of the auricle receives its sensory innervation through the auricular branches of the superficial cervical plexus, which also overlap the rim and supply a posterior marginal area on its external surface. The inferior and posterior walls of the canal are supplied by the small auricular branch of the vagus, which also sends filaments to the interior of the concha. The ganglionic representations of sensation on the auricle and external auditory canal have been divided between the Gasserian in front and the second and third cervical ganglia behind. The anterior half of this region has been referred to the Gasserian and the posterior half of the cervical ganglion. An eruption of herpes in this area has been regarded as emanating from disease of one or other of these ganglia.

The error of the prevailing views will be shown by a study of the anesthesia produced by the extirpation of these ganglia for the relief of tic douloureux. In all of Krause's cases following extirpation of the Gasserian the sensation of the skin of the auricle and external auditory canal was found to be preserved and normal. In these operations during the tearing out of the ganglion, the connections existing between the fifth nerve, Meckel's ganglion, the otic ganglion, and the geniculate ganglion through the superficial petrosal nerves may be separated. This might cause confusion, by adding a geniculate anesthesia to that produced by removal of the Gasserian. This may have happened in some of Cushing's cases in which parts of the external auditory canal were found anesthetic after extirpation of the ganglion. The method of operation as practiced by Frazier and Spiller is free from this disadvantage. It consists in cutting the sensory root of the fifth on the central side of the ganglion. In such a procedure as this there is no undue tension or tearing the neural connections between the geniculate, and the second and third divisions of the fifth are not implicated. No procedure could be more exact for protecting the ganglionic area

of the Gasserian. Following this operation the sensation of the auricle and auditory canal was found to be normal. The ganglionic innervation of the second and third cervical ganglia has been studied in a case of extirpation for the relief of obstinate occipito-cervical neuralgia by Harvey Cushing. In outlining the anesthesia in this case Cushing found the posterior marginal area on its external surface anesthetic. The interior of the auricle and external auditory canal had normal sensation.

If we now bring together and carefully adjust these respective areas of anesthesia, produced by extirpation of the Gasserian and the cervical ganglia, there still remains the interior of the auricle, and the external auditory canal in which sensation is preserved. In this area, I believe, it is to be found the cutaneous representation of the geniculate ganglion and its zoster zone.

It will be recalled that the peripheral innervation of this skin area is furnished by the auriculo-temporal branch of the third division of the fifth, and the auricular branch of the vagus. That these fibers do not pass through the inferior division of the fifth to the Gasserian is demonstrated by the anesthesia resulting from section of the sensory root of the fifth, this area retaining its sensation. If their afferent course is not through the trigeminus, how do they reach the brain? The relation of these sensory fibres to the geniculate ganglion is established if we accept the occurrence of geniculate herpes with a zoster zone in the auricle. So that it seems probable that these afferent fibres passing from the auricle on their way to the geniculate follow one or other of two routes, *i. e.*, from the skin of the auricle through the auriculo-temporal branch of the fifth, or the auricular branch of the vagus to the seventh nerve, the afferent fibres passing to the geniculate in the trunk of the facial nerve; or they may possibly be contained in the arriculo-temporal branch of the fifth to the otic ganglion and thence via the lesser petrosal nerve to the geniculate. Of these two routes, that through the facial is, in my opinion, the more likely one.

Clinical Types of the Disease.

Herpes Auricularis—The simplest manifestation is to be found in the characteristic and well-known picture of herpes zoster of the auricle. There are the usual slight prodromes in the initial stage of the infection, followed by fever and mild general symptoms. Then sharp, darting pains are felt in the ear, the preherpetic pains, sometimes reaching a high degree of intensity. The skin of the ear may assume a red, swollen, somewhat erysipelatous appearance, until on the third or fourth day typical patches of herpetic vesicles make their appearance. These are situated in the concha, on the lobule, the tragus, the marginal portion of the auricle (helix and antihelix), and within the auditory canal, indeed, as rarely happens, on the *membrana tympani*

itself. With the appearance of the eruption the acute pains usually subside, the ear still remaining swollen and tender.

At this stage of the orifice of the external auditory canal may become constricted by the swollen soft parts, so as to interfere with the proper drainage and cleansing of the canal. The defect in hearing which may result from this temporary occlusion of the meatus, is purely mechanical and is not in any sense related to the disturbances of audition which accompany another group of cases and which is dependent upon involvement of the auditory nerve. In a few days the vesicles desiccate, the swelling and edema of the parts subside until at the end of a fortnight only a few scattered zoster scars remain to tell the tale. The sensory symptoms may, however, persist for a considerable time; burning pains, itching, paresthesia with impairment of the cutaneous sensation of the parts. In old people more especially the sharp neuralgic pains in the ear, the post-herpetic pains, may persist for a considerable time. (Herpetic otalgia.)

In this class of cases it will be observed that the herpetic pains and the herpetic eruption are localized within that skin area which retains its sensation after extirpation of the Gasserian and the second and third cervical ganglia. It was this area which I assigned to the geniculate ganglion as its cutaneous representation and zoster zone.

Remarks—Idiepathic herpes zoster of the auricle has long been a recognized manifestation of zona. It is the herpes auricularis and herpes oticus of systematic writers. Dr. Anstie who was personally afflicted with the disease gave a very vivid description of his own case in "The Practitioner" of 1871. This localization of zoster has always been regarded as belonging to the trigeminal area, and due to disease of this nerve or its ganglion. If the observer favored the neuritic theory of herpes zoster, the skin lesions on the auricle were ascribed to a neuritis of the auriculo-temporal branch of the fifth nerve; if the ganglionic theory was accepted, the lesion was placed in the Gasserian. Some authors also speak of the auricular branches of the cervical nerves as playing a role in aural herpes, but no mention is made of the geniculate ganglion and its possible relation to this affection.

Compared with other manifestations of zona, the car is an infrequent localization. Gruber records five typical cases as occurring in a series of 20,000 cases of ear disease.

To determine, if possible, the relative frequency of these cases, I examined the annual reports of several of our large hospitals for the eye and ear, with rather varying results. The total number of cases recorded is surprisingly small, so small, indeed, that it seems very possible that the affection not infrequently escapes recognition. It may be that when seen in the early stage of intense inflammation cases are regarded as perichondritis or inflammation of the auricle, or when seen later after desiccation of the

vesicles, as cases of eczema of the auricle. In the Manhattan Eye and Ear Dispensary during the past ten years, with a total of 47,600 cases, the diagnosis herpes of the auricle was made in only two cases. In the Brooklyn Eye and Ear Hospital, during the past five years, with a total of 15,000 cases, the diagnosis was made but once. The New York Eye and Ear Infirmary averages at the present time 10,000 out-patients a year, and is one of the largest institutions of its kind in the world. During the past twenty-three years this diagnosis was recorded but six times. The reports of the Massachusetts Eye and Ear Infirmary show a much larger proportion of these cases. In the past ten years with a total of 65,000 cases, the diagnosis was made 33 times. In these tabulations it was not possible to determine whether the cases were of the true infectious type, or merely of secondary origin, but the infrequency with which the diagnosis was made is worthy of note.

Herpes Auricularis with Facial Palsy—In this manifestation of the affection, there is superadded to the herpes auricularis, as just described a peripheral facial palsy, which appears on the same side as the zoster. The time of the appearance of this palsy varies, coming on in some cases simultaneously with the eruption, in others it may be delayed a week or even longer. In the majority of instances it appears on the second or third day. Too much stress should not be given to the patient's statements in this respect as the onset is often insidious and unobserved.

The paralysis is complete and involves all three branches of the nerve, and has certain peculiarities. A conspicuous feature is the frequent evanescence of the symptom, evidences of paralysis lasting only a few days or a fortnight. Many of the palsies clear up within three weeks or a month. There still remains, however, a large group of cases in which the palsy is of a severe type, reactions of degeneration persisting for a long time, leaving permanent weakness and contractures of the face. It is also a striking fact that in an unusually large number of these cases the sense of taste is lost or altered. This is not surprising when one considers that the seat of the lesion is in the geniculate, a level where the taste fibres are still coursing with the facial.

I would explain the involvement of the nerve in this group of cases by the pressure of an inflamed and swollen ganglion or by the direct extension of the inflammation to the sheath and connective tissue structures of the nerve. In light palsies probably inflammatory edema and pressure are the factors at play, whereas in the more severe forms inflammation and structural changes probably take place.

Remarks—As has already been emphasized, a similar palsy may complicate herpes facialis and herpes occipito-collaris. I would explain the occurrence of the palsy in such cases by an herpetic inflammation of the

geniculate ganglion, based on the well recognized tendency of this affection to produce inflammatory changes in a series of spinal ganglia. The Gasserian, geniculate and upper cervical ganglia constitute such a serial chain.

These cases of facial palsy complicating herpes of the ear, face and neck, have long been the subject of study and controversy.

The old theories as to the origin of these palsies are as follows: The prevailing opinion is that the same exposure to cold produces both the herpes and the palsy, in which case the latter is regarded as rheumatic in nature, the common form of Bell's palsy. Another favorite theory was based on the infectious origin, the poison or toxin concerned in herpes zoster also producing a neuritis of toxic origin. A somewhat fantastic hypothesis which found great favor with certain observers was the following: The herpetic inflammation is supposed to have extended along the peripheral filaments of the trifacial nerve, this nerve having numerous points of inosculation with the terminals of the facial. The inflammatory process then passes by continuity of structure, directly from the peripheral filaments of the trifacial to the termination of the facial, in this way producing an ascending neuritis.

Herpes Auricularis with Facial Palsy and Auditory Symptoms—This is the most interesting as well as the severest type of the affection. In this group to the herpetic eruption on the ear, face or neck and facial palsy, are added symptoms pointing to involvement of the auditory nerve. The proximity of the terminal divisions of the auditory nerve to the facial and its ganglia, the common sheath and narrow osseous canal in which they lie would render such an auditory complication not only possible, but probable. Contributing factors may be severe forms of the inflammation or certain anatomical peculiarities such as a large ganglion or a narrow bony canal.

The auditory symptoms may be both irritative and paralytic in character and make their appearance about the same time as the facial palsy. First there is tinnitus aurium followed by progressive diminution of hearing. In the more severe cases the symptoms of Ménière's disease are also present. Disturbances of equilibrium, vertigo, nausea and vomiting, nystagmus. In the course of a few weeks the acute symptoms subside, the vertigo and disturbances of the gait and equilibrium disappear, but the tinnitus often persists for a considerable time and the hearing may be permanently impaired.

Remarks—In this group of cases as in that previously described the auditory symptoms may complicate herpes on the neck and face as well as on the auricle. In my series of 60 cases, auditory symptoms of various degrees of severity occurred in 19 cases. Of this number the zoster was in the occipito-cervical distribution in 9 cases, on the face in 4 cases, and on the auricle and auditory canal in 6.

The uncertainty which has attended the classification of this auditory group of cases and the doubtful nature of the affection, may be gathered from a perusal of the titles of the more important communications. "Ueber ein Fall von gleichseitigen, akut aufgetretene Erkrankung des Acusticus, Facialis und Trigeminus" (Kaufmann, 1896).—"Ueber Polyneuritis Cerebralis ménièriformis" (Frankl-Hochwart, 1899, and Berger, 1905).—"Zur Lehre von der peripherischen Facialis Lähmung" (Hoffmann, 1899).—"Beitrag zur Casuistik der multiplen Hirn Nerven Erkrankung" (Hammerschlag, 1898).—"Herpes Zoster Oticus" (Korner, 1904).—"Trouble Auditive dans le Zona" (Lannois, 1904).

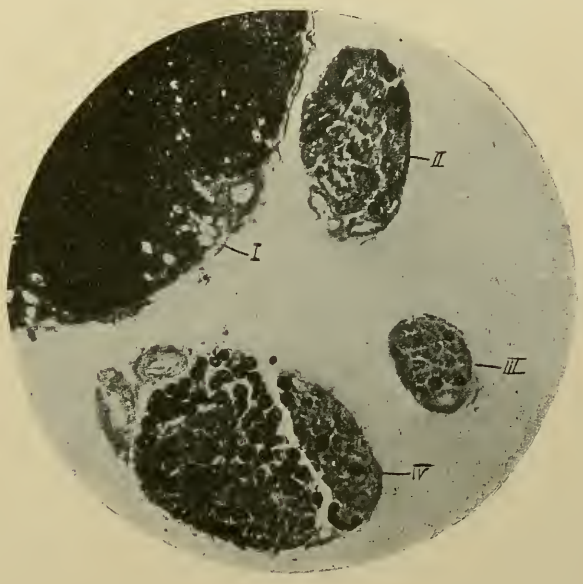

Fig. 5.

Osmic Acid Preparation, Counterstained with Rubin. I. Facial Nerve. II, III, IV. Bundles of the Nerve of Wrisberg, Showing Loss of Nerve Fibers and Sclerosis.

Pathological Evidence—Recorded below under Case II. is the pathological report of a case of herpes occipito-collaris in which a complete facial palsy supervened on the fourth day. There were no symptoms referable to the auditory nerve. Evidences of facial paralysis and objective sensory disturbances in the occipito-cervical region were still present at the time of death, which occurred 87 days after the onset of the disease. Corresponding to the cervical distribution of the herpes, old inflammatory changes were found in the tip of the third cervical ganglion, with loss of nerve fibers and islets of sclerosis in the corresponding posterior root of the spinal cord.

These could be traced through the 1st, 2d and 3d cervical segments, with evidences of myelin degeneration (granule cells and myelin droplets) in that portion of the posterior column, immediately adjacent to the postrior horns. Evidences of regeneration were also found in the branches of the suprficial cervical plexus. Such pathological changes, both in character and distribution, are in accord with the findings in similar cases.

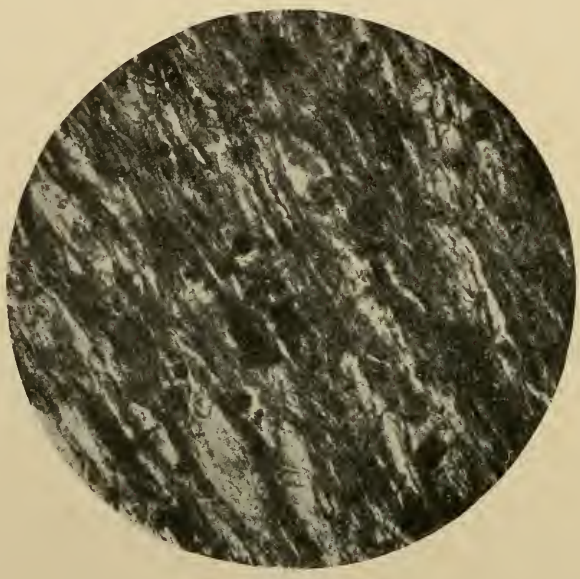

Fig. 6.

Marchi Method Intra-Medullary Degenerations in the Medial Root of the Acoustic. Entrance of the Nerve of Wrisberg.

It is, however, to the facial nerve that I would particularly direct attention. The facial nerve, including the nerve of Wrisberg, from its exit at the medulla to its entrance at the internal auditory canal, was treated by the osmic acid method and cut in transverse sections. By this method the nerve of Wrisberg was found to have lost a large number of its nerve fibers, with a compensatory increase of connective tissue. (See Fig. 5). Fortunately that portion of the medulla which was treated by the Marchi method corresponded to the roots of the facial, auditory and nerve of Wrisberg, all of these structures taking their origin at the same level.

The nerve of Wrisberg (*ars intermedia*) lies between the origin of the other two nerves, sometimes it joins one, sometimes the other, as it enters the substance of the medulla. Sections through this level show distinct evidences of degeneration (myelin droplets and granule cells) along the course of the internal root of the auditory nerve, after its entrance into the medulla.

(See Fig. 6.) In other words, we find in the nerve of Wrisberg, which is the sensory root of the 7th and having its trophic centre in the geniculate ganglion, the same extra- and intra-medullary changes as were found in the spinal cord and posterior nerve root corresponding to the 3d cervical ganglia.

Unfortunately that portion of the facial nerve removed and which was supposed to contain the intumescentia ganglioformis consisted only of membrane, so that the changes in the ganglion itself cannot be given. But even in the absence of the geniculate ganglion, the existence of well-marked degenerations in the nerve of Wrisberg and its intrabulbar root is sufficient proof that this structure was involved.

Case I. *Herpes facialis and occipito-cervicalis with facial palsy of short duration.*

A woman, aged 66, on March 20, 1904, was exposed for one hour to a sharp, cold wind. It was snowing at the time and the effects of the intensely cutting cold were keenly felt on the left side of the face and neck. Two days later sharp darting pains and tingling sensations developed in the left face, neck and occipital regions. These pains were soon followed by an eruption of herpes in the same distribution. Patches of vesicles were sparsely scattered over the skin area of all three branches of the trigeminus nerve, as well as in the occipital and cervical regions. The darting pains continued after the development of the eruption, until seven days after exposure and five days after the appearance of the vesicles, a complete left facial palsy was observed on awaking in the morning.

I saw the patient April 3, 1904, with Dr. Robert Denniston of Dobb's Ferry. At the time there was total paralysis of the left facial nerve in all its branches, with considerable sagging of the parts, lachrymation and dribbling of saliva from the corner of the mouth. Pressure over the 7th nerve in front of the ear caused pain. There were no demonstrable objective sensory disturbances of the left face or neck, and no tender points over the foramina of exit of the trigeminus nerve. Hearing and taste were not affected. There were no general cerebral symptoms and the pupils reacted normally. The other cranial nerves as well as the motility, sensation and reflexes of the extremities were entirely normal. The urine contained a slight trace of albumin but was otherwise negative. Heart sounds of good quality, no murmurs. One of the superficial cervical glands on the left side is slightly enlarged.

The sharp shooting pains continued for a week from this date, coming on usually at 1 a. m. and lasting three or four hours, and were very severe. They diminished in their intensity during the next fortnight and soon after disappeared. Within one month after the onset of the facial paralysis all

trace of it had disappeared, and the patient has since enjoyed her usual robust health.

Case II.—*Herpes occipito-cervicalis with severe facial palsy Autopsy and Histological Examination.*

The patient, admitted to the City Hospital February 15, 1905, was 48 years of age and a laborer. Periodic drinker. No venereal infections. The onset of the disease took place November 26, 1904, with sharp stabbing pains in the distribution of the right occipitalis major and immediately behind and in the right ear. With the sharp lancinating pains there was also present a burning sensation on the right side of the neck. These prodromal pains continued about four days and were followed by a vesicular eruption on the right side of the neck and the lower portion of the face above the ramus of the jaw. On the 3d of December, three days after the outbreak of the herpes a right facial paralysis was first observed. The vesicles dried up leaving many small pigmented scars. Paresthesias and burning sensations persisted with occasional sharp darting pains in the affected area.

Status Praesens, February 18, 1905—Complete right 7th paralysis of the peripheral type. Behind the right ear, along the ramus of the inferior maxilla and on the right side of the neck are scattered, pigmented post-herpetic scars. These are in the distribution of the 2d and 3d cervical segments. (Herpes occipito-collaris.) In this same area, sensations of touch, pain and temperature are diminished, especially over the angle of the jaw. Burning sensations and sharp neuralgic pains still persist in the scarred area. Taste and hearing are unaffected. The other cranial nerves, the pupillary and tendon reflexes are normal. Urine normal.

Electrical—To Faradism there is no response of the facial muscles on the paralyzed side when stimulated through the nerve. Directly applied to the muscles of the face the response is slow and very much diminished.

On March 12, 1905, developed fever with evidences of pulmonary consolidation of the upper and middle lobes of the right lung. Died March 26, 1905, at 5 p. m. Clinical diagnosis, tuberculous pneumonia.

Autopsy, March 28, 1905, at 3 p. m. A complete autopsy was performed by Dr. Oertel, pathologist to the City Hospital. The right lung was found to be the seat of an extensive tuberculous pneumonia. Gross examination of the brain and spinal cord was negative. Some of the cervical and dorsal spinal ganglia, branches of the superficial cervical plexus at its emergence from behind the sterno-cleido-mastoid muscle were removed for histological examination. The entire facial nerve from its exit at the medulla to its entrance at the porus acousticus, the facial and its geniculate ganglion in the Fallopian canal, both Gasserian ganglia and portions of the

pes anserinus of the facial were removed. The methods used were the Nissl, Osmic acid, Marchi-hematoxylin and eosin, Van Gieson and Weigert.

The Facial Nerve—Teased osmicised preparations of the *pes anserinus* show the presence of numerous fine pale fibers, with rosary-like nodulations and swellings of the myelin sheath. In many other fibers the myelin covering is thin and poorly stained (pale), showing intervals and interruptions in its continuity. There are no myelin droplets or granule cells and the whole picture is regenerative rather than degenerative. The tissue removed from the Fallopian canal and presumably consisting of the intumescentia gangliformis, unfortunatly did not contain this organ, so that a microscopical study of the geniculate was not possible.

The whole trunk of the facial and including the nerve of Wrisberg from its exit at the medulla to its entrance into the auditory canal was treated by the osmic acid method. Transverse sections were then made and counterstained with acid-rubin. By this method the fibers of the facial nerve proper were found normal. In the nerve of Wrisberg, however, there was demonstrated a very distinct and well marked loss of nerve fibers with a resulting sclerosis and increase of connective tissue. See Fig. 5.

Medulla Oblongata and Pons—The cells of the facial nucleus on the affected side under a low power are smaller, paler and more rounded than normal, with absence or only slight indications of the tigroid appearance of the Nissl bodies. Under a high power the nucleolus is but faintly stained, and the nucleus is small, irregular in outline and shrunken in appearance. In many cells the nucleus shows a partial or complete dislocation to the periphery. The Nissl bodies are broken up, the cell body presenting a finely granular appearance. In some cells there is the typical powdery appearance of axonal degeneration. The cell body is small, rounded and the processes broken off. The quantity and quality of the cell pigment does not seem to vary in the two sides. By the Weigert method the ascending root of the facial and the root fibers in the pons show no variation on the two sides.

Transverse sections of the medulla at the exit of the facial, the nerve of Wrisberg and the acoustic, show distinct evidences of old myelin degenerations, granule cells and myelin droplets in and along the course of the median root of the auditory nerve (Fig. 6). The root of the facial and the external root of the auditory are free from such evidences of degeneration.

Cord and Nerve Roots—The examination of the 1st, 2d, 3d and 4th cervical nerve roots on the affected side, shows distinct evidence of old degenerations in the posterior root of the 3d cervical segment. Delicate islets of sclerosis are demonstrable. These are quite destitute of nerve elements or contain only naked and swollen axis cylinders. No remnants of myelin degeneration were present in the nerve roots. The central tip only of the right 3d cervical ganglion was removed with the attached roots which

showed some thickening and proliferation of the connective tissue of the sheath on its internal surface. In the spinal cord distinct evidences of old myelin degeneration, myelin fragments, droplets and granule cells, were found scattered along the mesial side of the posterior horn and the adjacent white substance. These evidences of degeneration could be readily traced through the 1st, 2d and 3d cervical segments. The right Gasserian ganglion showed no evidences of inflammation or degeneration. The teased osmic preparations of the branches of the superficial cervical plexus, show the same regenerative changes as were described in the *pes anserinus* of the facial nerve.

Concluding Remarks—Briefly summarized my conclusions are as follows: The facial nerve like the trifacial is a mixed nerve. Its sensory ganglion is the geniculate. The motor root of the geniculate is the facial nerve proper and its sensory root is the nerve of Wrisberg. Below the ganglion the peripheral divisions are the facial nerve proper, the great and lesser superficial petrosal nerves, the external petrosal and the chorda tympani. This ganglion is of the spinal ganglion type and therefore in common with other ganglia of this type, comes within the realm of true herpes zoster.

The zoster zone for the geniculate is found in the interior of the auricle and in the external auditory canal.

The only neural connections existing between the geniculate ganglion and this cutaneous area are the auriculo-temporal branch of the 5th through the medium of the small superficial petrosal nerve and otic ganglion and through the facial nerve proper. One or both of these routes may be taken by the afferent fibers from the auricle in their central course; in my opinion the facial route is the more probable one.

The ear-zone of the geniculate is intercalated between the zone for the Gasserian in front and the cervical ganglion behind, so that the zoster zones of the cephalic extremity are represented by the Gasserian (face and forehead), the geniculate (ear), the 2d and 3d cervical ganglia (occiput and neck). The zoster inflammations while attacking chiefly one, not infrequently involve more than one ganglion, milder changes showing in a series of ganglia above and below, diminishing in intensity from the central focus. For the same reason zoster in any of the zones of the cephalic extremity, may be accompanied by inflammatory reaction in the other ganglia of this group.

The pathology underlying the affection is the specific hemorrhagic inflammation of the ganglion as found in zona. As the geniculate is lodged in a narrow bony canal and stands in close relation to the 7th and 8th nerves, the characteristic syndrome is produced.

This syndrome may be divided into three clinical groups:

1. Herpes zoster auricularis.

2. Herpes zoster in any of the zoster zones of the cephalic extremity (Herpes auricularis, herpes facialis, and herpes occipito-collaris) with facial palsy.

3. Herpes zoster of the cephalic extremity with facial palsy and auditory symptoms (Tinnitus, deafness, vertigo, vomiting, nystagmus and disturbances of equilibrium).

In the foregoing pages I have endeavored to outline as briefly as possible the anatomical, pathological and clinical facts upon which I have based the syndrome. For the sake of conciseness and clearness, I have eliminated as far as possible all material not absolutely necessary for a convincing argument. For the same reason but few direct personal references have been made to the work of the long list of able investigators, who were my predecessors in this field. In a subsequent communication I hope to be able to give the subject broader and more elaborate treatment.

Neuralgic Affections of the Geniculate Ganglion and its Divisions.

(Otalgia.) A Preliminary Report.

As the facial is a mixed nerve, having a sensory-ganglion, a cutaneous representation and zoster zone, the thought naturally arises, may it not be the seat of purely functional derangements similar to those occurring in the trigeminal area. May there not exist otalgia referable to the sensory mechanism of the facial, as there is prosopalgia originating in the sensory mechanism of the trifacial.

That herpes zoster of the auricle may be followed by severe neuralgic pains in the ear is well known. These herpetic otalgias are secondary, and in accordance with my views on this subject are dependent upon organic changes in the geniculate ganglion of the facial nerve.

On considering the extreme sensitiveness and delicacy of the organ of hearing, the high degree of its innervation, its exposed situation through the external auditory canal, the predisposition of such a mechanism to neuralgic disturbances would seem very probable.

This is the case. Otalgia, earache of non-inflammatory origin in its various forms is by no means an uncommon affection.

I would here mention briefly the branches and neural connections of the facial nerve and ganglion.

I. The ganglion stands in relation to the interior of the auricle and external auditory canal, its zoster zone (through the medium of the facial nerve or the small superficial petrosal nerve?).

II. The ganglion is intimately connected with the tympanic plexus. This is effected through the medium of the small and large deep petrosal nerves which arise in the tympanic plexus and join the small and large superficial petrosal nerves on their way to the geniculate.

III. The ganglion has also direct relations with the 2d and 3d divisions of the 5th, through the medium of the great and small superficial petrosal nerves which pass to Meckel's and the Otic ganglia respectively.

IV. It has reflex connections with the glosso-pharyngeal nerve through Jacobson nerve, which enters into the formation of the tympanic plexus.

V. Another reflex connection is with the vagus through its small auricular branch. This nerve sends an ascending and descending filament to the facial as it crosses the Fallopian canal.

From these brief statements an idea may be obtained of the intimate neural relation existing between the facial nerve and ganglion and the tympanic cavity, auricle and external auditory canal; as well as the reflex neural connections with the trigeminus, glosso-pharyngeal and vagus nerves. A great many of the otalgias are of reflex origin. In these forms there occur with caries of the teeth and ulcerative affections of the tongue, pharynx and larynx, neuralgic pains, localized in the ear. These cases are quite common and the relation between the nerves supplying the region of the mouth and naso-pharynx and larynx with sensation and the tympanic cavity and auricle furnish an anatomical basis for the reflected pain.

The primary otalgia, idiopathic neuralgic affections of the ear, are recognized by nearly all systematic writers. This group of cases, however, as is the case with auricular herpes is divided between the trigeminal and occipito-cervical nerves. If the pain predominates in the anterior half of the auricle, they are usually relegated to the trigeminal, or if in the posterior to the occipito-cervical neuralgia. I believe, however, that there exists an otalgia, an independent primary and idiopathic affection of the sensory system of the facial nerve, and that this group will occupy the same place and have the same significance as the other time-honored clinical group of neuralgias.

MULTIPLE NEURITIS: THE RESULT OF POISONING BY CYANIDE OF POTASSIUM. CLINICAL AND EXPERIMENTAL.

A Contribution to the Toxic Effects of Cyanide of Potassium Upon the Peripheral Motor Neurons.

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The peripheral motor neurons, that is, the ganglion cells in the anterior horns of the gray matter of the spinal cord, and their processes, particularly the neuraxons or axis-cylinders, are susceptible to injury by many toxic and infectious agencies. When such agencies affect the cell bodies or intra-spinal processes of the cell-bodies, the resulting disease is known as poliomyelitis. When the extra-spinal processes, or neuraxons, are diseased, the condition is called neuritis. For a long time it was thought that the same agency which produces disease of the cell-body was not likely to produce disease of the neuraxon, and in a measure that belief is founded upon fact. The agencies that are most prone to cause inflammation of the motor nerves constituting motor neuritis, are probably not those responsible for inflammation or decay constituting poliomyelitis, save perhaps in very exceptional instances. Numerous investigators have shown that certain metallic substances, such as lead, causes degeneration of the motor nerves constituting a true motor neuritis. Such cases coming to autopsy have shown distinct degeneration of the anterior horn cells of the spinal cords. Moreover, it has been abundantly shown by many investigators, particularly by Stieglitz (Arch. f. Psych., vol. 24, p. 1), that experimental lead poisoning produces degeneration of the entire motor neuron. The question which awaits reply in those cases is the same as we shall raise in the present article, *i. e.*, which part of the primary motor neuron is first and principally affected in certain intoxications?

We have been unable to find any cases of neuritis or poliomyelitis produced by cyanide of potassium poisoning in a fairly careful search of the literature. This article is offered as a contribution to that subject.

We report herewith a case of acute inflammation or degeneration of the peripheral motor neurons, which constitutes clinically a peripheral motor neuritis, but which, however, can scarcely be distinguished from an anterior

poliomyelitis. In fact, so far as the reporters of the case are concerned, it cannot be distinguished.

The patient, an Italian, 38 years old, was admitted to the City Hospital December 3, 1906. A brief synopsis of the history of his illness is as follows: Since coming to this country, in 1904, he had been working as a silver polisher in a hotel. The method of keeping silver bright in such establishments is to drop it into a solution of cyanide of potassium and then dry it. He says that his hands and forearms were in such solution so much of the time that they took on a deep brownish red color and he frequently complained of a distressing itching sensation in them. In addition to this the finger-nails were quite black. He does not know how strong the solution of cyanide of potassium was that he used, but he says that he had to be careful to keep his hands away from his nose or his mouth, for otherwise he would get very dizzy. On the 7th of September, 1906, he was seized with diarrhoea, which soon became very severe, the passages containing large amounts of mucus, but no blood. The following day he complained of severe headache, of pain and stiffness in the back of the neck and of feeling ill. These symptoms continued for four or five days. Then there developed a sensation of stiffness of both ankles and severe pain in the legs below the knees. Within 48 hours his legs and arms were so weak that he could scarcely move them and from that time he was unable to stand or walk. About this time, *i. e.*, eight days after the onset of his symptoms, he had retention of urine and had to be catheterized for more than a week. After that he had difficulty in expelling the urine, but he got on without having a catheter passed. At this time he did not complain of pain or parasthesia, the symptoms were entirely of the motor apparatus, nor were there any trophic symptoms apparently. His best recollection concerning the atrophy of the muscles, which was so conspicuous on his entrance into the hospital, was that it was first noticed about eight weeks after the onset of his disease.

Examination at this time showed that the patient was bed-ridden, unable to move the lower extremities and to perform very few movements with the upper extremities. The atrophy of the muscles was the most conspicuous objective symptom, and the distribution of this atrophy was as follows: The trapezii were involved to a slight extent. The supra and infraspinati muscles, the serratus magnus, the deltoids, the biceps to a very considerable degree, and the triceps very conspicuously atrophied. The plantars and the extensors of the forearm were also very much shrunken. In the lower extremities the muscles chiefly involved were the anterior tibial group and the posterior calf muscles, the vasti and quadriceps. The left leg lies in a position of external rotation, and it is impossible for him to rotate it inwards. Both feet are in a position of drop-foot, the right being more

profoundly so than the left. The dorsal surfaces of the feet are slightly cyanotic; the nails show transverse ridges at their bases. The knee- and ankle-jerks are absent. There is a flexor plantar jerk. There is marked atrophy of the glutei over the left hip. In the upper extremities the left deltoid and the trapezius are atrophied and this atrophy throws the shoulder lower than on the opposite side. There is some atrophy of the muscles of both forearms, and there is marked shrinkage of the thenar and hypothenar eminences of the left hand. (See Figs. 1 and 2.)

Tactile, thermol and pain sensibility are normal. There is no disorder of function of any of the cranial nerves.

Electrical examination shows that the anterior tibial muscles contract on both sides with a strong faradic current. There is a slight response to the faradic current in the quadriceps, but the reaction is sluggish and vermiform. In the other atrophied muscles the faradic contractility is lost apparently in proportion to the intensity of the atrophy. In all the muscles there is a slight reaction to the galvanic current with polar alteration characteristic of reaction of degeneration.

For upward of six months there seemed to be no indications of recovery. Then gradually the atrophied and paralyzed muscles of the upper extremity began to display slight functional capacity. In the autum of 1907 braces were put upon his legs and now he is able to walk with the aid of crutches. Examination of him at the present time shows that the muscles of the neck and trunk are of good volume and capacity. Both deltoids are atrophied, the left more profoundly than the right. External rotation of the shoulders is weak. The muscles of the arm and forearm seem to be in good condition, save the supinator longer and the biceps of both sides. The triceps, which was very much affected in the beginning, now seems to be approximately normal. The extensors of the right wrist are weaker than those of the left, but there is no evidence of atrophy in the muscles.

The cranial nerves are intact.

Flexion of the thighs is accomplished very indifferently, but better on the left side than on the right, while extension of the legs upon the thigh, which is also very weak, is accomplished better on the right than on the left. There is double foot-drop. He can flex the toes of the right foot but not of the left. The crural muscles and the calf muscles show the greatest amount of atrophy.

The knee-jerk is present on both sides but extremely weak and sluggish. The abdominal and cremasteric jerks are elicitable. The right Achilles jerk is present but sluggish, and the left absent. Tactile, pain and thermal sensibility seem undisturbed. There is no tenderness of the nerves to deep-seated pressure. Distinct fibrillary movements are occasionally to be seen in the gluteal and deltoid muscles.

In other words, the patient has made a fairly good recovery and he is still improving. The only symptoms that tend to indicate the dependence of some of the atrophy upon spinal cord lesion, is the fibrillary phenomenon to be observed in the muscles in which the atrophy is still most pronounced.

Taken in conjunction with the experimental work that has been done to determine the effect of cyanide of potassium upon the peripheral motor neuron, it is obvious that the entire motor neuron was affected by the poison, but that the brunt of the lesion was borne by the axons at their periphery.

The important features of the disease as they presented themselves in this case were: (1) The rapidity with which the symptoms came on; (2) the occurrence of spinal cord symptoms; (3) distribution of the atrophy and partial recovery.

Unlike the neuritis that is caused by other metallic poisons such as lead and arsenic, there seem to have been no premonitory symptoms whatsoever, although there are cases of lead neuritis in which there has been no history of colic, digestive disturbances, etc., and there are cases of arsenical neuritis which are not preceded by the peculiar garlic odor of the breath; such cases are very uncommon. In our patient there seemed to have been no premonitory symptoms except the symptoms of gastro-intestinal irritation. The extent and intensity of the atrophy as well as the intensity of the motor incapacity which preceded it, bespeak the profound involvement of the peripheral motor neurons.

From the experimental work which we have undertaken to show the effects of potassium cyanide upon the paripheral motor neurons, we are led to the belief that the entire motor neuron is affected, the neuraxon, however, more than the cell-body, but the participation of the cell-body in the destructive process is fairly certain, and this participation accounts for the occurrence of spinal cord symptoms in our patient. That the atrophy, however, that is, the degeneration in the muscles, is not primarily dependent upon spinal cord lesion, is best testified to by the distribution of the atrophy. It would have to be an extremely extensive process in the spinal cord and such extensive lesion would cause symptoms that did not occur in this case. Moreover, the degree of recovery which has taken place indicates that the cell-bodies of many of the peripheral motor neurons, which at first seemed to be very profoundly diseased because of the amount of atrophy, are in reality in possession of sufficient vitality to assist in the regeneration of the diseased neuraxons.

The following experiments were done for the purpose of studying the effects upon the nervous system of cyanide of potassium administered in poisonous doses:

Four healthy rabbits were used in the experiment. The two first died, the two last recovered.

The salt of hydrocyanic acid used was the potassium salt, which was made in a sterile water solution, 1 c.c. equaling 2 mg. of the pure salt. The injections were made with a sterile hypodermic syringe, usually into the posterior auricular vein. The wound was immediately sealed with collodion.

All spinal cord specimens were hardened in 10 percent. formalin, followed by alcohol, embedded in celloidin and cut at about 6 microtons; sections were then stained in a 1 per cent. aqueous blue, being generally heated until the dye steamed; they were then differentiated in absolute alcohol, 90 parts, and anilin oil 10 parts, washed in alcohol and cleared in xylol.

Rabbit No. 1 received 2 mgs. of the drug every other day for a period of sixteen (16) days, or a total of 16 mgs. On the sixteenth day a complete flaccid paralysis of both hind legs was noticed, accompanied by loss of flexes, and bladder and rectal incontinence. Sensation was apparently normal. On the seventeenth day the rabbit was much worse; both forefeet were now completely paralyzed, and there was slight twitching of the neck muscles. On the eighteenth day, as the animal lay in his cage, he was totally helpless, unable to hold up his head, and paid no attention when food was offered. Sudden disturbances, such as noise, were followed by convulsive movements of the head. Towards evening he died, sixty (60) hours after the appearance of paralysis.

The following is a summary of the autopsy findings:

Dilatation of the left ventricle of the heart; pulmonary oedema, with hemorrhage in bronchi; parenchymatous degeneration of kidneys.

The following tabulates the distribution of the lesion found in the spinal cord: At the level of the oblongata, the anterior horn-cells were normal. Sections from the upper portion of the cervical enlargement were also normal; sections from the mid-portion of the cervical enlargement showed some neurons in various stages of degeneration. Sections from the mid-dorsal showed marked destruction of the anterior horn-cells of both horns. Sections from the lower dorsal also showed extreme destruction. Sections from the lumbar region showed scattered cells in various stages of destruction. Sections from the extreme lower lumbar region showed apparently normal neurons.

The changes observed in the anterior horn-cells were central chromatolysis with nuclear eccentricity, leading to entire destruction of cytoplasm, with vacuolization, shrinkage, and almost complete solution of the cell.

The lesion apparently commences in the chromophilic bodies which do not stain with the same intensity as they normally do; they appear pale and have ragged edges. At the same time, the nucleus also becomes swollen, and its nuclear membrane becomes well marked and distinct. (Fig. 3.)

The chromophilic bodies now become granular and disintegrate, causing a great diminution in the number of bodies; this takes place first around the

axone hill and nucleus. The cytoplasm around the nucleus becomes vacuolated, filled with fissures, or even entirely devoid of matter.

While the chromatolysis is going on, the nucleus also shows changes. Owing to a loss in substance of cytoplasm, it is left unsupported and falls to one edge of the cell, it becomes eccentric. The hyleoplasm becomes granular, causing the nuclear membrane to become puckered and crenated. The nucleolus remains clear and sharply defined, it being retained when all other structures of the cell are lost. The nuclear membrane is finally lost and complete destruction of the nucleus soon follows.

With such great loss in bulk of cell, it, as a whole, shrinks, leaving an open cell space; the cell may finally break up and disappear. (Fig. 4.)

A portion of the right anterior tibial nerve, when teased out in glycerine and stained with osmic acid, showed extensive degeneration, with destruction of the myelin sheath, and the presence of irregular size fat droplets.

Rabbit No. 2 received 2 mgs. every other day for ten (10) days. At the end of this time the animal became very quiet, respirations and pulse were rapid, food was refused. On the morning of the next day the animal was paralyzed in both hind legs; at noon his forefeet were involved, and he died during the night, about twenty-four (24) hours after paralysis had started.

The spinal cord was taken out immediately and studied, with the result that the anterior horn-cells, throughout the mid-cord, and in the cervical enlargement, showed the various stages of chromatolysis and neuron degeneration above described. Teased portions of the anterior tibial nerve showed fatty degeneration of myelin sheath. (Fig. 5.)

Rabbit No. 3 received 12 mgs. over a period of fifteen (15) days. Except for slight diarrhoea it had no ill effects.

Rabbit No. 4 received as an initial dose 4 mgs. He immediately went into severe convulsions with rapid panting respirations, which soon stopped. Artificial respiration revived the animal from the acute cyanide poisoning. After this he received a total of 14 mgs. in fourteen (14) days, but no paralysis developed.

In the two last rabbits many of the injections were not made directly into the vein, but in the surrounding subcutaneous tissue.

There has been a great deal of controversy as to the nature of chromophilic bodies. F. B. Bailey (Journal of Experimental Medicine, 1901), concludes that "Nissl bodies are not present in the living cell, and that the chromophilic bodies seen in the Nissl preparations represent a precipitation of the substance previously held in suspension in the cell protoplasm, caused by post-mortem change, or action of fixatives." These bodies are best observed in tissues fixed in acid fixatives.

It is also not certain that the cytoreticulum is present in the living cell (as it can be seen only after fixation), or whether it, too, is a precipitate formed before, or at the time of the formation of the Nissl bodies.

These observations, however, make little difference in the case in question, as we know the chromatolysis (as described by Nissl), is not the rule in the normal neuron, and that a disappearance of the chromophilic bodies, as seen in these cases, associated with vacuolization and nuclear disintegration, is caused by a neuron injury, which leads to a partial or complete degeneration.

In conclusion, it would appear that the changes observed in the anterior horn-cells were due to a further extension of a peripheral multiple neuritis, that is, an axone degeneration which is so severe that it finally affects the neuron itself. In a chromatolysis, due to toxic material in the blood serum, which bathes the periphery of the cell, the chromophilic bodies are first destroyed around the periphery of the cell, and they last disappear around the nucleus, or, in other words, a peripheral chromatolysis is more common than a central one. A nuclear eccentricity is also very uncommon. In these cases, the foregoing, however, is not true. Here we have a chromatolysis which takes place, first, around the axone hill, and around the nucleus (a central chromatolysis), indicating that the cell has suffered internally, so to speak, by the extreme degeneration that has already taken place in its axone.

FIG. 1.

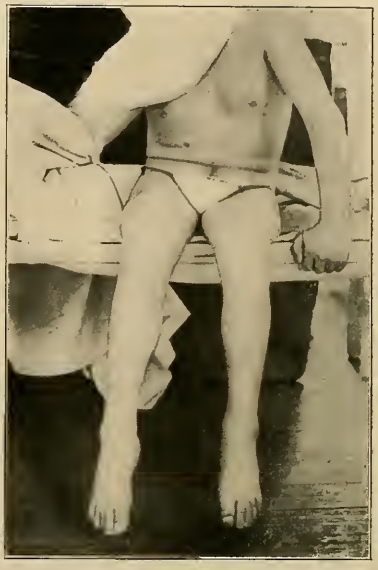

FIG. 2.

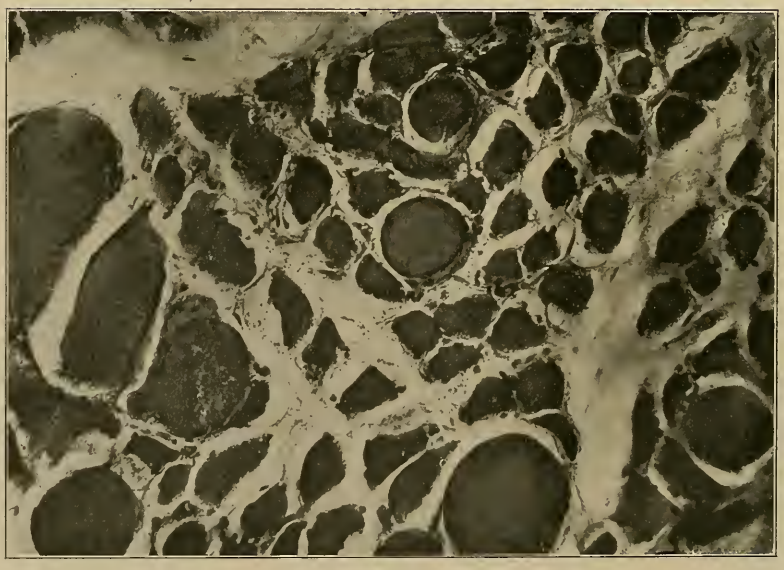

FIG. 3.

Section from deltoid muscle of patient (harpooned). 300 dia. Showing parenchymatous degeneration. Note variation in sizes of muscle bundles; the large, homogeneous, hyaline bundles; the small, wasted bundles which are the seat of numerous minute granules. The perimysium around the small bundles is relatively prominent.

FIG. 4.

Section of Anterior tibial nerve from Rabbit No. 1. 300 dia. Osmic acid stain. Showing extensive parenchymatous neuritis or degeneration, with presence of small and large myelin droplets in the medullary sheath, and granular detritus. The axis cylinders are the seat of swelling, vacuolization and disintegration.

FIG. 5.

Section of Anterior tibial nerve from Rabbit No. 2. 300 dia. Osmic acid stain. Showing a beginning parenchymatous or neuritus with commencing segmentation of the medullary sheath, and slight coagulation of myelin between the indentations. The axis cylinders are swollen and turbid.

VI.—Dermatological Division

THE VESSEL CHANGES AND OTHER HISTOLOGICAL FEATURES OF CUTANEOUS SYPHILIS.*†

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While the literature covering the pathologic anatomy of syphilitic lesions is very extensive, it contains little which tends to elucidate the manner in which the process develops and spreads, many of the descriptions applying equally well to the other infectious granulomata. In order to arrive at any satisfactory conclusion or obtain a proper conception of the reaction the syphilitic organism produces it is necessary to examine an enormous amount of material, not only in different stages of evolution of a given lesion, but also in series. The present article is an endeavor, not by any means to cover the entire histopathology of cutaneous syphilis, but rather to point out some features in the histologic picture of the early and late lesions which are more or less pathognomonic and which perhaps offer some data on which to base a microscopic diagnosis.

Aside from the presence of the spirochaetae in the tissues, the reaction which they excite is more or less peculiar to this condition and should enable one to make a diagnosis in the majority of instances. It is a not infrequent experience of dermatologists to meet with cases where surgeons have excised initial lesions on extragenital parts under the mistaken diagnosis of malignant growths. To the expert the clinical features of such lesions should be sufficiently distinctive to exclude the latter, and where there is a doubt it is often a simple matter to examine histologically and differentiate the growth from carcinoma, which is about the most common condition which comes into question.

Hoffmann[1] believes the spirochaetae enter the lymph spaces of the rete where they are protected against phagocytosis or immediately make their way into the lymph vessels of the papillae. Only after about three weeks, when multiplication has taken place, does a reaction appear. The first changes then would be an endo- and perilymphangitis, followed by budding of capillaries, infiltration and connective tissue hyperplasia. According to the findings of Ehrmann[2] the lymph vessels are involved secondarily, the earliest change taking place in the connective tissue interstices, followed by new capillary formation and perivascular infiltration. He based his deductions on gelatin injection experiments with initial lesions immediately after

* The material from which these studies were made was obtained chiefly from patients in the City Hospital.

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their excision. Unless special technic is observed the vessels can not readily be differentiated. In a general way, however, the arteries are distinguished from the accompanying veins and lymphatics by their thicker coats and greater abundance of elastic fibers. In an infiltration the media is relatively free and appears as a band separating the intima from the exudate. The walls of the lymphatics are thinner than those of the veins. The lymphatic capillaries are apt to have a more irregular lumen than the blood capillaries and a less complete lining. Their lumina often contain infarctions of a homogeneous substance and sometimes a few lymphocytes.

In practice, as a rule, patients do not present themselves until some days after the appearance of the primary sore, when it has already progressed beyond the classical coat-sleeve infiltration and both blood and lymph vessels are involved. The following are brief descriptions of several chancres, all with the same characteristic changes but in different stages of development:

In one, less than a week old, the epidermis was eroded over a portion of its extent and compressed into a narrow band with obliteration of the pegs. Beyond the erosion acanthosis and emigration of leucocytes were the chief changes. In the corium the infiltration was quite diffuse superficially, encroaching upon the epidermis, but below it was distinctly "coat-sleeve" (Fig. 1), and consisted of small lymphocytes, plasma cells, mast cells and proliferated fibroblasts, many of the latter being uncommonly long, either spindle-shaped or stellate with fine processes. Large cells also appeared with irregular bodies, granular protoplasm and hyperchromatic nuclei, some eccentric and others central; they were probably cross sections of swollen fibroblasts. Where the cells were confined to the vessels they were several layers deep and consisted of the round and plasma variety. The capillaries were increased in number and, with the exception of a few in the upper cutis which were dilated, the majority had either swollen or proliferated endothelium. The walls of the larger vessels were thicker than normal and their lumina somewhat narrowed. A number of the lymph spaces were simply dilated, others showed proliferated endothelium. There was no apparent collagenous hypertrophy or edema.

In a specimen six days old the epidermis over the most active part of the lesion was converted into a flattened amorphous layer containing many round and polynuclear cells and, superficially, blood pigment. The ridge system was replaced by a dense zone of cells, lymphocytes predominating. On either side the contiguous epidermis showed acanthosis and emigrated leucocytes. In the cutis the area of greatest intensity was made up of many new-formed capillaries and an infiltration of lymphocytes, plasma cells, mast cells and fibroblasts. A number of occluded blood and lymph capillaries were scarcely recognizable in the dense exudate; others had swollen or

FIG. 1.

FIG. 2.

FIG. 1.—Chancre of the penis. (Zeiss 8mm., comp. oc. 4.) An early lesion with mantle of round and plasma cells about vessels. The latter are increased in number and show an endothelial proliferation. The intervening connective tissue contains hyperplastic fibroblasts and a few plasma cells and lymphocytes.

FIG. 2.—Chancre of the penis. (Zeiss 8 mm., comp. oc. 4.) An older lesion still showing the ensheathing of the capillaries with lymphocytes and plasma cells, but with the infiltration more diffusely distributed through the remaining tissue. All the vessels have a proliferated lining. In the center is seen a longitudinal section of a capillary.

proliferated endothelium and had their lumina distended with blood or narrowed as the result of proliferation and compression. The larger vessels showed a decided thickening and infiltration of the adventitia, with or without a corresponding change in the intima. The connective tissue of the cutis was the seat here and there of a slight fibrosis.

In an ulcerated chancre there was a great increase in capillaries in the upper layers of the corium, some of which were filled with blood; others were empty. The infiltration, of the same type as in the preceding cases, was so dense that the cells had the appearance almost of a mosaic. Longitudinal, oblique and transverse sections of the capillaries showed proliferation of their lining and narrowing or obliteration of their lumina (Fig. 2). The larger vessels in addition were the seat of a periarteritis and infiltration. In the inferior portion of the cutis were present several large vessels containing organized thrombi, their walls presenting the characteristic changes of a panarteritis. There was no appreciable hypertrophy or edema of the collagenous tissue.

In a chancre at the height of its development there was an acanthosis of the epidermis, with clubbed and knobby rete pegs, some edema and parakeratosis. The cutis was very cellular, only a few strands of connective tissue being visible here and there. A narrow band in the inferior portion of the cutis had the appearance of condensation. A number of blood and lymph capillaries in the papillary layer were dilated and empty, with no apparent change in their endothelium. In those situated more deeply, however, an endarteritis obliterans was present and only solid cords remained to indicate their previous position. Many of the vessels contained thrombi of a homogeneous material which partially or completely filled their cavities. Evidences of fatty degeneration and retrograde metamorphosis were present in patches of cloudiness and vacuolization.

In sections from the edge of a fully-developed chancre, including healthy tissue, were seen some acanthosis of the epidermis, the pegs being more widened than lengthened, and an invasion of leucocytes. In the papillary and upper reticular layers of the corium through the edge of the chancre the infiltration was diffuse and the blood and lymph capillaries were markedly dilated and increased in number, some containing thrombi. Endothelial proliferation was slight, the cells being more closely packed but not encroaching on the lumen. Passing through the reticular layer of the hypoderm the infiltrate was very dense and many of the cells so distorted from pressure that it was difficult to say to which type they belonged. In this area no lumina were visible, only the thickened occluded vessels. In the adjoining tissue an endo- and periarteritis were present and the perivascular exudate was composed largely of plasma cells. Bands of hypertrophied

connective tissue surrounded some of the vessels and part of the active infiltration.

Summing up, then, the main features of the primary lesion we have: 1, An increase in blood vessels and consecutive changes in their walls; 2, cellular infiltration; 3, involvement of the connective tissue; 4, secondary epidermic changes.

Many causes have been assigned to the hardness of the sclerosis, as capillary lymphangitis with suffusion of the neighboring connective tissue;[3] infiltration cells, thickening of the vessel walls, new growth of connective tissue and dryness and rigidity of the cutis; hypertrophy of the collagenous tissue;[4] swelling of the collagenous tissue, distention of lymph spaces with lymphocytes and an edema.[1]

Of those that have come under my observation only a few showed fibrosis and, with the exception of two, in so slight an amount that it does not seem sufficient of itself to produce the degree of hardness clinically appreciable. The two referred to are the last one above described and a flat chancre in which hyperplasia was a noteworthy feature. In another lesion of the chin, which was very hard clinically, the sections were almost entirely cellular, with numerous vessels and but little connective tissue, so that the collagenous tissue hypertrophy as the factor which has been evoked by Unna and others does not seem to explain the condition. The closely aggregated sclerosed vessels probably play a greater role than has been given them.

The erosion is analogous to the central involution of the later lesions and is due to necrosis of the infiltration and the overlying epidermis. In phagedenic chancres one or several elements are to be considered; either secondary infection, lack of resisting power on the part of the affected individual, vessel changes and possible toxins capable of producing local necrosis.

The involution of a chancre is brought about by fatty degeneration, the compression and narrowing of the vessels leading to disturbances in nutrition and retrograde changes. Ehrmann is of the opinion that spontaneous healing takes place by phagocytosis.

When it comes to secondary manifestations of the disease, as a rule, all doubt is removed and a histologic examination as an aid to diagnosis does not come into question, excepting, perhaps, in certain drug eruptions, as bromid and iodid, which may simulate very closely certain ulcerative and fungating syphilitic lesions. A case of this sort recently came under my observation, where fungating lesions were present about the face and genitals of a young woman along with a general rash, which were distinguished with great difficulty from a possible specific infection. Fungating growths of the vulva resembling flat condylomata were examined very carefully for spirochaetae with negative result.

The evolution and histology of the roseola and papule seem to show that these lesions are embolic rather than toxic. A possible explanation of the development of circinate and gyrate lesions by the involvement of a terminal vascular cone has been suggested.

In the roseola Hoffmann[1] found spirochaetae in the dilated end capillaries of the papillae, and the long interval which elapses between their entrance in the body and the appearance of the efflorescence suggested that the organisms give off few toxins, which only after a longer period are capable of producing a deleterious effect. Or, according to Thalmann, an endotoxin liberated on destruction of the body may be causative.

Histologically, a macular lesion presents a mild reaction. The epidermis may be unchanged or flattened out with loss of the ridge system. In the upper corium the vessels were moderately dilated, filled with blood and ensheathed by a slight infiltration of round cells. The endothelium was swollen, and round and spindle cells were seen in the adventitia of the larger vessels. The cutaneous appendages were normal and surrounded by a few lymphocytes.

The small miliary papule, lichen syphiliticus (Fig. 3), is a follicular process extending from the epidermis into the cutis below the hair papilla. The infiltrate was located about the hair follicle, which remained intact and sent outgrowths into the surrounding nodule, which nodule was composed of a reticular structure holding new-formed blood vessels, round, plasma and connective tissue cells in its meshes. Most of the vessels inside of the nodular area were obliterated, but at the periphery they were filled with blood. A number of the superficial ones were thrombosed and appeared like giant cells. Away from the perifollicular disposition the vessel walls were thickened somewhat and mantled by round and plasma cells. The coil glands were frequently the seat of a marked fatty degeneration and surrounded by an effusion of round and plasma cells. The erector pili muscles were likewise infiltrated. The epidermis was slightly edematous and thinned. There was present sometimes a pustule, produced by the instability of the tissue matrix and liquefaction of the syphilitic infiltrate.

In the large papular syphilide there were present in the epidermis parakeratosis, acanthosis and edema, with a marked invasion of polynuclear leucocytes. Epithelial mitoses were rather frequent. The cellular infiltration in the early stages (Fig. 4) of the lesion was limited to the vessels in the upper part of the corium, and the papule was produced chiefly by the epithelial hyperplasia, but later it was more diffuse and surrounded all the vessels of the deep and superficial plexuses. Most of them had thickened walls and consequent diminution of their caliber, but here and there a few remained dilated and showed little if any change. Some were partially thrombosed. Not infrequently immediately about some of the vessels and

Fig. 3

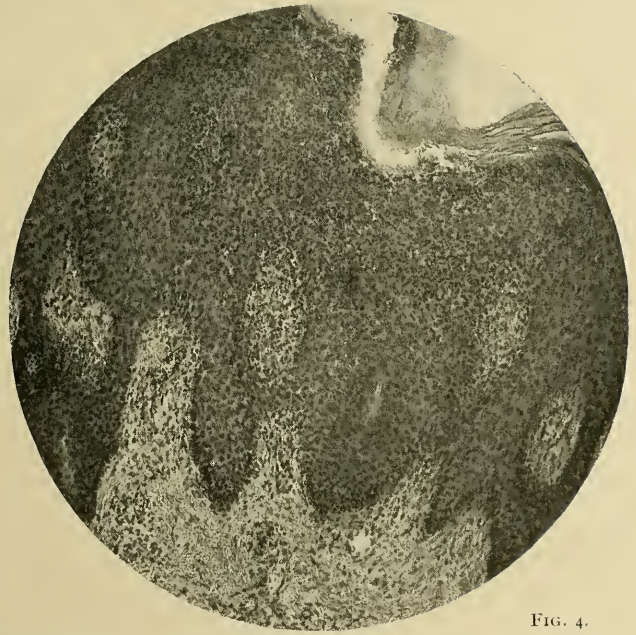

Fig. 4.

Fig. 3.—Small papular syphilide, *Lichen Syphiliticus*. (Spencer 1 in., Zeiss comp. oc. 4.) Illustrating the follicular nature of the lesion. The process extends along the hair follicle into the cutis below the hair papilla and consists of a reticular structure with round, plasma and connective tissue cells in its meshes. Inside the area many of the vessels are completely obliterated; others are thrombosed and simulate giant cells. At the periphery of the infiltration a few engorged vessels are present. Away from the perifollicular part the vessels are thickened and sheathed by a few round and plasma cells. The coil glands are surrounded by the same cellular elements and are the seat of a marked fatty degeneration. Two outgrowths from the affected hair follicle penetrate the active process. The epidermis presents no noteworthy changes.

Fig. 4.—Papular syphilide. (Spencer ½ in., Zeiss comp. oc. 4.) A lenticular papule removed in the early stage of the disease, showing acanthosis of the epidermis, with dense infiltration of leucocytes and necrosis of the upper layers. In the papillary bodies there is slight sheathing of the vessels with round and plasma cells. The lesion is produced chiefly by epidermic hyperplasia.

coil glands there occurred a degeneration of the connective tissue, reducing it to a fibrillary network which took the basic stain faintly and was probably toxic in origin. The cellular exudate was composed of round cells with a few plasma cells scattered between them. There was also hyperplasia of the fibroblasts. The pustulation which occurs in the large papule represents in a major degree the central involution which takes place.

The rupial syphilide showed a very marked parakeratosis with leucocytic infiltration of the entire horny layer and here and there an aggregation of cells, coagulated serum and detritus entering into the crust formation. The overgrowth of the rete was irregular, many of the pegs being narrow and pointed, others widened and bulging into the cutis. It was edematous, in places necrotic, and lymphocytes were so numerous in it that many of the epithelial cells were completely hidden.

The cellular effusion in the corium was quite superficial, being almost limited to the papillary and immediately subjacent layer, except where it followed along the course of a hair follicle or mantled a few of the deeper vessels and coil glands. In the papillae blood and lymph capillaries were markedly dilated, some containing a homogeneous substance. Where the inflammatory reaction was most intense the vessels were narrowed, the larger and deeper ones showing more of an adventitial proliferation. The fibroblasts were increased in number; no giant cells were present. A mucinous degeneration appeared about the coil glands and some of the vessels.

In the nodular or tubercular syphilide the entire cutis from the epidermis to the subcutaneous fat tissue was involved, under a low power showing alternating light and dark areas, the former representing the altered blood vessels and the latter a cellular effusion. Under the high power the infiltration was composed of plasma and giant cells, lymphocytes and hyperplastic fibroblasts. The vessels, increased in number, showed the effects of an obliterating endarteritis (Figs. 5 and 6), their former site being marked by solid cords, groups of irregularly-disposed cells with pale staining nuclei and giant cells with peripheral or central nuclei or both. Near the edge of the section were a few vessels with narrowed lumina and a hyperplasia of all their coats. A number of the lymph spaces were thrombosed. The few remaining bundles of connective tissue had here and there a vacuolated appearance, the result probably of a fatty degeneration and beginning regression. The epidermis was flattened out into a very thin band.

The gumma (Fig. 7) compromises the derma throughout its entire extent with marked vascular changes, a diffuse infiltration and necrotic foci. Viewed with the low power, there was apparently a decrease in the number of blood vessels, but on careful examination it was found that many were obliterated or had their caliber reduced and their walls thickened. The infiltration consisted of lymphocytes, plasma cells and a large number of

hyperplastic fibroblasts. One or more areas of caseation were observed and about them fragmented nuclei. When intact the epidermis usually presented no changes beyond a reduction in thickness.

From the foregoing descriptions it will be seen that the differences existing between the various specific lesions are of degree rather than kind. With the exception of the macular syphilide, all consist of a dense infiltration of the same kind of cells, as a rule the lymphocyte being the most common. However, in some specimens lymphocytes, in others plasma cells predominate, but not with sufficient constancy to accept their presence, numerically, as of pathognomonic value. The exudate begins in the papillary layer about the superficial plexus, from thence involving the deeper tissue.

Changes in the collagenous tissue, except as it is replaced by granulation tissue, are not regular and are often so slight that they are not worthy of comment. In viewing a series of lesions from a given number of cases I found that ordinarily the changes were not sufficiently well marked to be of any practical diagnostic aid. Nevertheless, both hypertrophy and atrophy of the connective tissue, as a result of vessel changes, do occur. An instance of the latter was reported by me in a case of symmetrical cutaneous atrophy with the coincident development of syphilis of the skin and nervous system,[5] the alterations in the vessels' walls (Fig. 8) and the perivascular infiltration pointing strongly to these structures as the primary seat of the trouble. Or a productive interstitial inflammation may be secondary, which is illustrated in the accompanying photomicrograph (Fig. 9) of a syphilitic laryngitis, the larynx having been extirpated under the mistaken diagnosis of a malignant growth.

It is not uncommon to find an epithelioma developing on a syphiloma (Fig. 10), especially about the oral cavity. The epithelial hyperplasia is probably brought about by irritation, increased hyperemia, as well as by changes in the underlying connective tissue permitting an epithelial downgrowth.

Coming to the vascular system, in all the lesions except the macule the reaction is marked, being of greatest severity in the late secondary and tertiary stages of the disease and showing all grades from swelling of the endothelium to proliferation of all the coats and obliteration of the lumen. It is very likely that there are two processes, one taking its origin in the adventitia and the other in the intima, possibly simultaneously, or, as investigations on the cerebral arteries suggest, independently of each other.

Hansemann[6] is of the opinion that the syphilitic virus has a special predilection for the endothelial cells, whether of the endocardium, large or small arteries, veins or lymphatics; all the rest may be involved secondarily. To the vascular changes may be attributed the formation of giant cells so frequently encountered in the nodular and tubercular syphilide and in some

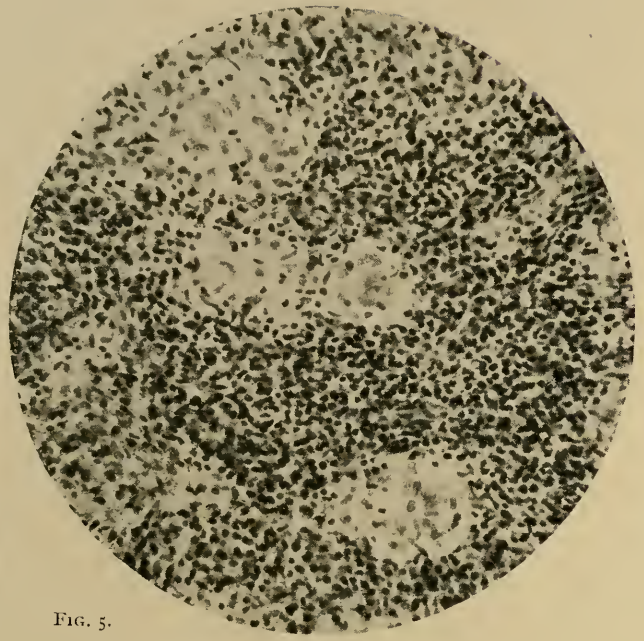

Fig. 5.

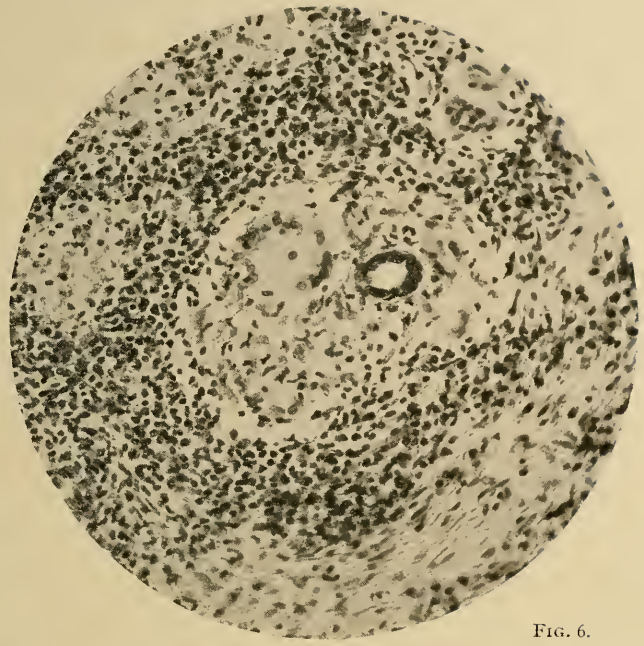

Fig. 6.

Fig. 5.—Tubercular syphilide of the face. (Spencer ¼ in., Zeiss comp. oc. 4.) Showing obliteration due to endothelial proliferation and mantling of the transformed vessels by round and plasma cells.

Fig. 6.—Tubercular syphilide. (Zeiss ¼ in., comp. oc. 4.) A section showing obliterated and thrombosed vessels with a perivascular infiltration of round and plasma cells. A number of the vessels show simply an endothelial proliferation. The one in the center is, in addition, thrombosed and simulates a giant cell.

FIG. 7.

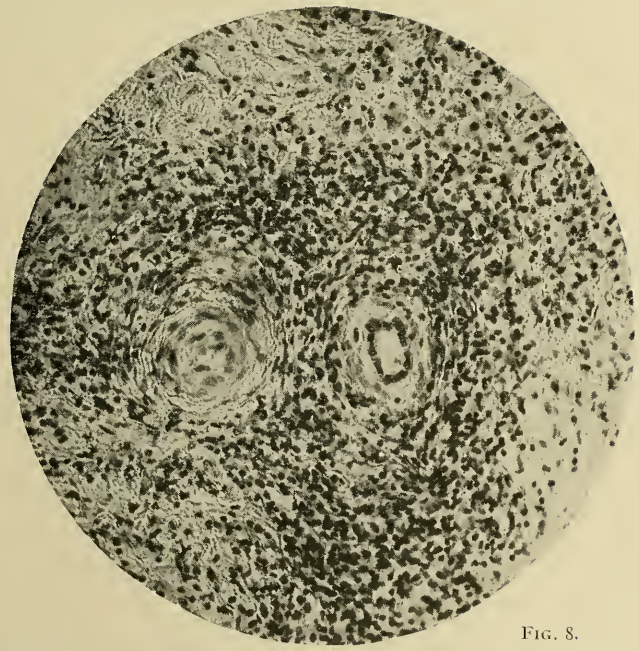

FIG. 8.

FIG. 7.—Miliary gumma of the skin. (Spencer 1 in., Zeiss comp oc. 4.) The cutis is uniformly infiltrated with round, plasma and proliferated connective tissue cells and here and there shows areas of caseation. One is seen in the illustration in the middle third of the corium just to the right of the lmedian ine. The vessels have their caliber reduced in size or are entirely obliterated. The epidermis is thinned and its pegs are flattened out.

FIG. 8.—Symmetrical atrophy of the skin with syphillis. (Spencer ¼ in., Zeiss projection oc. 2.) Cross sections of an artery and a vein. The former shows a proliferation of its intima and adventitia with narrowing of the lumen; the latter is completely occluded by intimal proliferation. Surrounding them is an infiltration of round and plasma cells.

of the early secondary papules. It is rare to find them in gummata, and it has not been my experience to find them in initial lesions, although Unna and Rieder claim to have done so. They appear to be a more constant feature of the tubercular and serpiginous lesions, in which they are often present in groups. In many instances it is simple enough to trace their formation to proliferated endothelium and thrombosed lumina. In others it is much more difficult to establish such a relationship, but if sections in series are studied it is demonstrable. The type most frequently met with is the Langhans with peripheral nuclei; the myeloid with central nuclei occasionally. In one case a double row of nuclei appeared whose formation it was difficult to explain.

Giant cells of specific lesions differ from those found in lupus in being less completely formed, in showing in many instances only a partial thrombosis and in their irregular nuclear multiplication, suggesting that the process is a more acute one. As they are more numerous in serpiginous than other lesions, it has occurred to me that the extension of those lesions could be explained by the progressive thrombosis of the vessels. Many specimens support this idea, but especially one from a serpiginous lesion of the thigh (Fig. 11), in which the histologic findings consisted of foci of groups of thrombosed vessels with a moderate cellular effusion.

While apparently true of cutaneous syphilis, it is not intended to impute to metamorphosed vessels the origin of giant cells in all specific processes. Versé[7] found in arteritis syphilitica cerebralis giant cells in all layers of the vessel wall in one of his cases, but especially in the intima where they flanked the entrance to an aneurism and had an enormous number of nuclei. In other cases they were found in the necrotic media. He believed that many of them were foreign body giant cells. v. Baumgarten[8] has expressed the belief that the presence of giant cells in syphilitic processes indicated a combination of tuberculosis and syphilis, but in none of the cases that came under my observation was tuberculosis demonstrable anywhere.

The syphilitic products show a greater instability in some lesions than in others, as in the pustular, rupial and gummatous. In the two former, perhaps, the question of soil is the more significant, while in the latter the severe vessel alterations interfering with nutrition offer an explanation. The involution of the efflorescences is brought about by necrobiosis, fatty metamorphosis and caseation.

Recurrences in pre-spirochaeta days were explained by Neumann[3] as due to the proliferation of cells which remained at the site of the original lesion. Now, however, the multiplication of remaining spirochaetae is invoked as the cause by Neisser.[9]

An important aid in the clinical diagnosis of syphilitic skin lesions is often rendered by the observation of the so-called pigmentary syphilide of the neck, which is occasionally seen either as a primary condition or as a

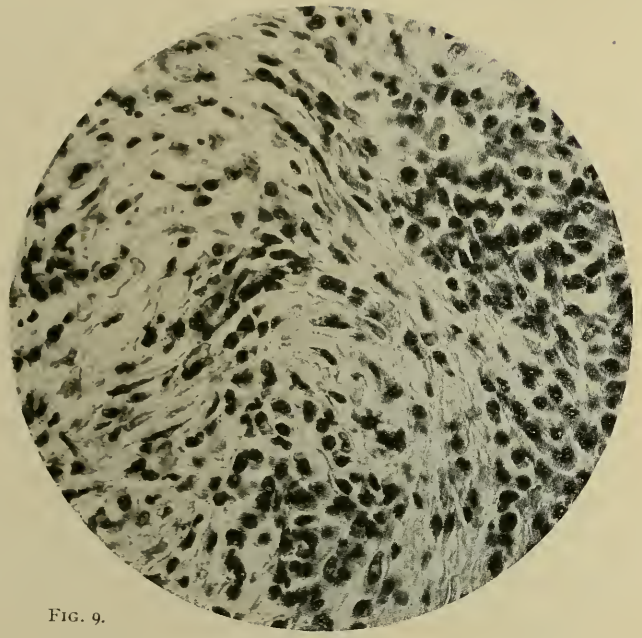

FIG. 9.

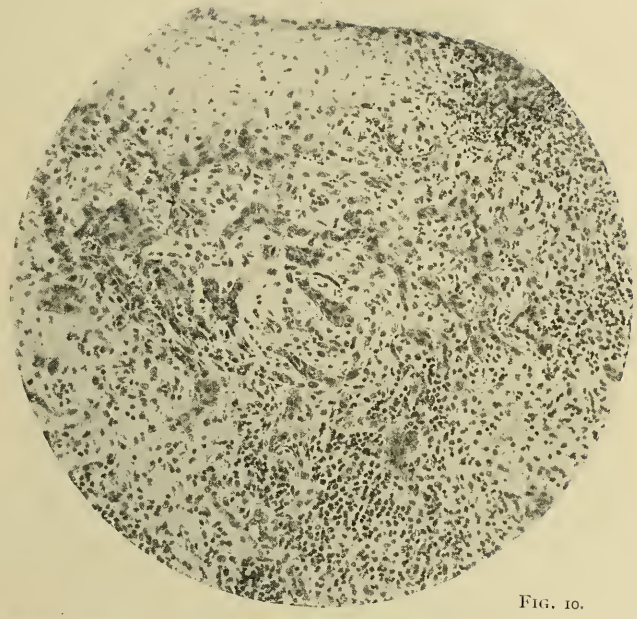

FIG. 10.

FIG. 9.—Syphilitic laryngitis. (Spencer ¼ in., Zeiss comp. oc. 4.) A productive inflammation of the larynx, showing hyperplasia of the connective tissue and an inflammatory infiltration of round cells and large numbers of plasma cells. The vessels are the seat of an endarteritis or panarteritis.

FIG. 10.—Epithelioma developing on a syphiloma of the lip. (Zeiss 8 mm., comp. oc. 4.) One portion of the lesion shows an ulceration extending to muscle of the lip. It is the seat of an intense infiltration of lymphocytes, through which are scattered some plasma and many polynuclear cells. There is a marked increase in the blood vessels and some hemorrhage is present. A number of the vessels are dilated and filled with blood, their walls presenting no perceptible change. Others are simply thrombosed, or again, their walls are thickened and the lumina occluded by proliferated endothelium. Several giant cells with peripheral nuclei are traceable to vascular changes. At the edge of the lesion are seen fine processes of proliferating epithelium penetrating the derma.

FIG. 11.

FIG. 12.

FIG. 11.—Serpiginous syphilide of the thigh. (Spencer ¼ in., Zeiss projection oc. 2.) A group of vessels giving rise to giant cells, one of which shows very distinctly the double row of nuclei. The surrounding infiltration is composed of round, plasma and connective tissue cells.

FIG. 12.—Late syphilide of the nose simulating hypertrophic lupus. (Spencer ¼ in., Zeiss projection oc. 2.) A section through an area containing a group of capillaries which have been completely obliterated and present the appearance of giant cells. The connective tissue is increased in volume and pervaded by fibroblasts, round cells and plasma cells.

sequel to a pre-existing erythematous or papular eruption. It occurs more often in brunettes and is an important corroborative evidence of syphilis after other manifestations of the disease have disappeared. Pigmentation in syphilis is favored by a cachectic condition of the individual and also by the presence of an old nephritis. In my service at the City Hospital it is not infrequent to see a syphilitic infection in a patient with an antecedent nephritis, in whom the development of pigmentation is a common occurrence and often very marked.

Recently Hoffmann[1] has ascribed to the spirochaetae a role in the diminution and increase of pigment in specific lesions. He found that in the areas invaded by the organisms the pigment in the epidermis was entirely lacking or very much reduced in amount. This depigmentation he considers the result of a local action—the body toxins—of the spirochaetae. The deeper marginal pigmentation surrounding spots of leucoderma is probably brought about by their peripheral progression, which occasions at first an increase and later, by intensity of action, a disappearance of pigment. The diffuse bronzing of the skin, seen especially about the neck, he believes to be due to the action of the spirochaetae on the suprarenals, for which they appear to have a special predilection.

The conditions from which syphilis is to be differentiated are tuberculosis, lichen scrofulosorum, blastomycosis, leprosy, epithelioma and drug eruptions. It often requires the minutest examination to draw a distinction between specific lesions and lupus, as exemplified in several specimens in my collection, notably a syphilide from the flush area of the face (Fig. 12), and another from the back of the finger. The latter had been diagnosed clinically as a lupus. The following points may serve as a guide: In syphilis there is a greater degree of proliferating endarteritis and other vascular changes; many of the giant cells are incomplete and can be traced to the vessels, and progressive and retrogressive changes are more rapid than in lupus.

The small follicular lesion presents a picture so closely resembling that of lichen scrofulosorum that it can scarcely be differentiated from it microscopically, and we are obliged to take into consideration the clinical features, as the age of the patient, the presence of concomitant symptoms of syphilis and the absence of scrofulosis.

In blastomycosis and leprosy it is such a simple matter to demonstrate their respective organisms in the tissue that the histologic changes are of secondary importance. Epithelioma is readily excluded by the presence of an epithelial growth in the corium.

The most frequent drug eruption that comes into question is the bromid, the lesions of which present epithelial hyperplasia with miliary abscesses and a diffuse infiltration of round and polynuclear cells, often with

eosinophiles through the derma. Giant cells and vascular sclerosis are lacking.

All clinicians have observed that certain specific lesions yield to treatment with great facility, while others are obstinate in their response to therapeutic measures. It does not seem possible from ordinary histologic examinations to offer any explanations or draw any conclusions as to why this is the case. It may be that in one the spirochaetae are more malignant and in another the resisting power of the patient lowered. On the other hand, the reason may be sought on anatomic grounds, the implication of certain structures, as the vascular apparatus, for instance, where proliferation may interfere with the permeability and absorption of the therapeutic agent. However, these are simply hypothetical questions for which as yet no satisfactory solution is offered.

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A CASE OF UNDETERMINED TROPICAL ULCERATION INVOLVING THE NOSE, PHARYNX AND LARYNX, WITH HISTOLOGICAL FINDINGS.*

By John A. Fordyce, M.D.

Attending Dermatologist.

AND SOME GENERAL CONSIDERATIONS REGARDING CLINICALLY SIMILAR CASES IN OCEANIA AND ELSEWHERE.

By W. F. Arnold, M.D.

Surgeon U. S. Navy (Retired).

The case, which forms the subject of the following report, was under the observation of my colleagues and myself at the City Hospital during a period of about eight months, and gave rise to a considerable degree of interest because of the unusual clinical picture which it presented. A diagnosis of syphilitic ulceration, chiefly from the appearance of the pharyngeal and laryngeal ulceration and cicatrization, was at first made and active anti-luetic medication was employed without in any way influencing the process. Mercury was given internally, by inunctions and hypodermatically, and iodide of potassium in increasing doses until the limit of tolerance in more than three hundred grains daily was reached. The failure of this treatment to favorably influence the condition strengthened the opinion held by several gentlemen, who saw the patient, that the ulceration was due to tuberculosis. Repeated examinations of smears and sections of excised tissue failed to demonstrate the presence of tubercle bacilli. Three guinea-pigs were inoculated with scrapings from the ulcerated surface. Two of them, inoculated in the peritoneal cavity, died within thirty-six hours of general septic infection, one showing a pronounced peritonitis and the other a double pleurisy. The third guinea-pig, inoculated subcutaneously, showed no symptoms of general infection, but within three months a superficial ulceration appeared, extending from the root of the tail, where the inoculation was made, over the back and causing a loss of hair. The patient in the latter part of March, 1905, received two injections of tuberculin (two and four milligrammes, New York Board of Health) at intervals of forty-eight hours, without causing the slightest reaction. The absence of tubercle bacilli in the sections and tissue, with a negative result from the inoculations and a failure to respond to the tuberculin test, seemed to effectually dispose of the provisional diagnosis of tuberculosis.

* Reprinted from the Journal of Cutaneous Diseases, Vol. XXIV., 1906, page 1.

It was then thought that the process might be a form of tropical ulceration, which was contracted while the patient resided in Panama. This view was strengthened by reading the report of Breda [1] on nasal ulcerations in Italian emigrants returning from Brazil, to which he gave the name framboesia brasiliana or boubas. A short resume of Breda's paper is given by Jeanselme,[2] of which the following is a partial translation:

"The affection is distinct from syphilis and yaws and begins by a bulla or pustule, which gives rise to a remarkably indolent ulceration with sharply defined infiltrated margins and a gray nodular base. It involves not only the skin, but even more frequently the mucous surfaces, especially the vault of the palate, the throat, the larynx and the trachea. The voice is profoundly modified early in the affection. Anti-syphilitic medication proved unavailing and no reaction took place after injections of Koch's tuberculin."

In some of its essential features, the malady described by Breda corresponded to the case under consideration and forcibly suggested that we had to do with a tropical ulceration of unknown nature, of which the clinical features had been recognized by the Italian observer. The strongest reasons for regarding the disease as a distinct entity were given me by Dr. Arnold, as the result of his experience in the Island of Guam, and, at my request, he kindly united with me in the preparation of this paper.

The patient, J. B., colored, aged forty-four, native of Panama, was admitted to the City Hospital in the autumn of 1904, with the diagnosis of lupus of the nose and larynx.

Family history—his parents were both dead; his father was shot in war and his mother died of old age. No history of insanity, tuberculosis or malignant disease was obtained.

Past history—occupation: he worked on a rubber plantation. He did not remember any childhood diseases. He suffered considerably from malaria while a resident in Panama, but had no periods of prolonged illness. Five years ago he had an attack of gonorrhoea with no complications. He had no urinary symptoms except some slight dribbling following expulsion of urine. Syphilitic infection was denied.

Present history—he came to the hospital complaining of nasal obstruction and ulceration, causing continual muco-purulent discharge, bleeding, pain and difficulty in breathing. It began six years ago when he was in Panama, the first symptoms being a very annoying and offensive muco-purulent discharge from his nose and a gradually increasing difficulty in breathing. For a period of six months he allowed it to go on as he was in the woods gathering rubber and no physician was at hand. Then he returned to Colon and immediately sought medical advice. A polyp was

removed, but the operation instead of giving him relief, only seemed to aggravate the condition, for his symptoms increased and the process extended. A year later small ulcers appeared about the nasal orifices. The patient stated that about three years prior to the beginning of his trouble, he was engaged in gathering rubber, which necessitated climbing trees on which grow vines called in Spanish "peca behuca," because the milky juice burns the skin wherever it touches. The English translation is "Bay Sore." By some people this sore is credited to the water drunk. At any rate, he insisted that when the hatchet cut the vine the expressed juice caused a peculiar raised somewhat circular sore with a central root (?). Of these he had two which appeared simultaneously, the one the size of half a dollar on the radial side of the left forearm just above the wrist, the other, about two and one-half by three inches in diameter, on the back of the right forearm just below the elbow. They lasted about three months and were eventually cured by an ointment of unknown nature, which, according to the patient, burned somewhat and took the "water and the roots" out of the sore. They showed no tendency to recur, and although he continued this occupation for years, he had no more of them. He mentioned that during the years 1876-1877 these sores were very common among the inhabitants of Costa Rico and the coast and the people often had ulcers about their nostrils, with resulting deformity of the nose.

Physical examination—colored, rather emaciated. Nose very broad and ulcerated at external meatus; tip sunken.

Lungs—dullness over both apices, more marked over right. Percussion was not very good over anterior or posterior surfaces of chest. Vocal fremitus poor. Vesicular murmurs good and full, but prolonged and high pitched at both apices, especially at right apex behind. At right apex there were some crepitant and many moist and sibilant rales. At left apex and over rest of chest there were many fine and coarse mucous rales. Voice sounds loud over right apex.

Heart—precordial dullness from mid-sterum to within nipple line. Apex not seen; felt in fifth space within nipple line, not very forcible. A slight systolic murmur at apex was transmitted a little to the left. The other sounds were muffled, except the aortic which was accentuated. The arteries were thick and stiff. Pulse was small and feeble.

Liver—dullness from the fifth space to just below costal margin, where edge was felt one finger lower down.

Spleen not felt through abdomen.

Eyes—pupils equal, reacting to light and accommodation. Sclerae yellowish-white and injected. Just below right inner canthus an ulcerated patch one by one and one-half centimeters; borders raised, center necrotic, a bloody serum exuding on pressure.

Nose—very broad at tip with great ulceration around edges of nostrils, involving a part of the upper lip (Plate I); septum gone and the entire anterior nares one necrotic sloughing mass with hardly any recognizable landmarks.

Mouth—pharynx very pale; uvula gone; soft palate presented a worm-eaten appearance.

Larynx and pharynx showed old cicatrical contractures with here and there ulcerating areas. The cords were rough and reddened and did not approximate. The arytenoid bodies were red and enlarged. The tongue was atrophied at the base.

Arms—there was an extensive but not very deep scar on the left forearm, and a scar from a burn on the right arm near the wrist—pigmented but not extensively contractile. Numerous tattoo marks on both arms.

Legs—knee jerks and plantar reflexes were present. Some old scars, white and pigmented, on anterior surfaces.

Urine—specific gravity 1022, alkaline; no albumen, sugar or casts.

From September 28 to October 19 his temperature varied from 98.2° to 101.2°, the mean being 99.5°. He was discharged on the latter date, but returned on November 7. On that day he had a severe chill which lasted twenty minutes and his temperature rose to 103.8°. Between that date and December 17, on other days there was a fluctuation of 1°-1½°. He was discharged again and returned on March 22, when he had another chill lasting about five minutes, followed by a temperature of 101.8°. From then until April 8 there was a variation between 98° and 99.6°.

The most striking feature of the foregoing history was the involvement of the mucous membranes and underlying tissues of the nose, pharynx and larynx by a chronic ulcerative process, apparently distinct from tuberculosis and syphilis, which began in the nose and later extended to the larynx and possibly to the trachea or even to the bronchi. The photograph (Plate I) shows only the later extension of the disease to the anterior nares, upper lip, the right nasal duct and lachrymal sac. The edge of the ulcer on the upper lip was marked by sharply defined margins and elevated edges and showed only slight enlargement during the time the patient was in the hospital. Unfortunately, he demanded his discharge and passed from observation so that the ultimate result of the disease could not be determined. As to the prognosis of the affection, the literature of carefully observed cases is too scanty to permit us to draw very definite conclusions. Among the patients under Breda's observation, although the skin lesions healed, new foci recurred for years on the mucous surfaces of the nose and upper air passages. The tendency of the disease, too, as shown by the cases seen in Guam, is to progressive involvement of new areas of

skin and mucous membrane, to loss of vision from destruction of the eyes and finally, to complete obliteration of the features.

While the clinical picture presented some striking features of syphilis, the long duration of the disease in a limited area, and its failure to respond to anti-syphilitic remedies, would seem to exclude that infection. The involvement of the nasal duct by extension of intra-nasal syphilis, though seen once in the writer's experience, is certainly an unusual occurrence in that disease.

Histology.—The tissue examined microscopically consisted of two pieces removed with a cutaneous punch at the junction of the anterior nares and lip. One was from the advancing margin of the lesion, the other from the central and older part. They were fixed in alcohol and Müller-Formol, respectively, embedded in paraffin and cut serially. Tissue stains employed were Böhmer's haematoxylon with orange G and with eosin, Unna's polychrome-methylene blue and glycerin ether, methylene blue and eosin, Van Gieson, Weigert, Unna-Taenzer orcein and Unna's collagen stains. To demonstrate possible organisms the following were used:

The Gram method, which revealed only a few positive cocci: the Ziehl-Neelsen, the Gabbet and the Koch-Ehrlich technic for tubercle bacilli, with negative results; carbol-thionin, Nicollé, likewise proved negative. In connection with this stain, it might be mentioned that a misleading precipitate occurred in the form of rod-like bodies bearing a close resemblance to organisms, which was only correctly interpreted after making control preparations of other lesions. Fungous or protozoan forms were not demonstrable in any of the sections examined with that end in view.

In the sections from the diseased edge one-half of the surface was ulcerated, the other showed a proliferated epidermis. The former was covered with blood and fibrin, underneath which there was a dense cellular infiltration extending to the muscle tissue of the lip (Plate III). The cells consisted largely of lymphocytes of the small variety. Only a few polynuclear leucocytes were present and plasma cells varied in number throughout the series. They were especially numerous in the deeper parts of the cutis about the vessels in the neighborhood of the muscles. The same may be said of the mast cells which also were larger than ordinarily and contained coarser and a strikingly greater number of granules. Numerous granules were free in the tissue and at some distance from the cell. In addition, there were epithelioid cells and hyperplastic fibroblasts. The giant cells were irregularly distributed; in some fields one or more were found close to the epidermis, again they were in the reticular layer and even in the subcorium. They were large, with one or more rows of peripheral nuclei and the origin of some could be traced, serially, to the blood vessels. While the role of the latter in true giant cell formation is disputed, and

Baumgarten maintains that such appearances merely result from sections through thrombosed vessels, it is conceded that a minute examination of some of the cells strongly suggested the latter; but others, apparently, were produced by a proliferation and fusion of the endothelial cells, while still others defied all effort to establish a genetic relationship with any of the elements present. Whether they are true giant cells or not, their likeness in some instances to those of other granulomata was so close that it was impossible to make the distinction. Search for foreign bodies was futile, and as noted above, no micro-organism excepting a few cocci were brought to view with the stains enumerated.

The reticulum of this granulation tissue was very delicate in places and only here and there small remnants of elastic and collagenous tissue remained. There was no cheesy degeneration. Below where the cellular invasion was less marked, the fibrous and muscular structures were normal. Throughout the area there were many blood vessels, newly formed and old. The larger ones were distended, their walls showing more or less inflammatory changes, while the majority of the smaller ones were in a state of partial or complete obliteration, owing to a thickening of their intima. Of the lymph spaces, many had entirely disappeared, others were dilated or filled with proliferated endothelium. There was also evidence of hemorrhage in the tissue.

The appendages presented no abnormality beyond a dilatation of the coil glands.

In the deepest layers of the hypoderm, surrounded by extravasated blood, was a small area whose component elements consisted mainly of lymphocytes with some fibroblasts and plasma cells in a fine reticulum. The vessels in this situation were narrowed or occluded by a proliferated endothelium.

The epidermis at one end was divested of its horny covering; at the other toward the ulcerated region a parakeratosis and acanthosis existed, with a tendency for the pegs to lengthen, widen and to form an anastomosing network (Plate IV). Some of the prickle cells were vacuolated and leucocytes had invaded the various layers.

The sections from the older portion of the lesion agreed in their essential features with those from the margin. There was, however, a greater vascularity, many dilated and filled vessels occupying the papillary and subpapillary layers and the infiltration was more nodular in its character. Below the diffuse superficial zone there were three distinct foci, which did not differ in their architecture from the others except that they contained more giant cells (Plate V). The intact epidermis was also the seat of an oedema and the interpapillary processes passed as long fine strands into the underlying structure and gradually became lost among the foreign cells.

In June the guinea pig developed a folliculitis which first appeared near the site of inoculation and spread up the back to his neck. The hairs fell out, some of the lesions became confluent and three ulcerated areas resulted. The course of the affection was about four weeks, during which time the animal seemed entirely well; there was no adenopathy and no loss in weight. A piece of tissue excised showed a rather simple superficial ulceration covered by a crust of blood and detritus. The infiltration in the corium below was made up chiefly of polynuclear leucocytes and lymphocytes (Plate VI). Of the various bacterial stains, only Gram showed a few cocci in the crust. Scrapings from under the crust gave no growth on ordinary media.

With so little evidence it is difficult to say, if the condition was a parasitic one, whether the pig became infected by chance after the inoculation, or whether the causative organism was introduced at the time of the injection. If the latter, the incubation period was a long one—over two months. Healing was spontaneous and at present not a vestige of the disease remains. The hairs have all come in again except over two of the deeper ulcerated areas, which are replaced by scar tissue.

Summary.—The lesion was a granuloma whose nature could not be determined by the methods employed.

Briefly, it could be differentiated from others of its class, like blastomycosis, actinomycosis, rhinoscleroma and leprosy by the absence of their specific micro-organisms. From mycosis fungoides by the character of the infiltrate and the absence of fragmentation. The histological picture might readily be mistaken for tuberculosis, as the giant cells were numerous with nuclei arranged peripherally, and as is often seen in that type of inflammation, many independent foci containing three or four such cells were encountered deep in the corium. The epidermic hyperplasia was such as is met with in hypertrophic lupus and other forms of skin tuberculosis; but as it is seen also in blastomycetic dermatitis and other cutaneous inflammations, its presence is of slight diagnostic importance. The existence of a tuberculosis, however, would seem to be conclusively disproved by the failure of the inoculation and tuberculin tests, as well as by the absence of bacilli in the secretions and sections.

It is also probable that the affection was distinct from yaws, as such vascular changes, giant cells, small number of leucocytes and great disintegration of fibrous stroma are not characteristic of the latter. It is not unlikely that many of the so-called tertiary manifestations of yaws are in reality not due to that disease, but to another infection identical with the one under consideration.

It was difficult to exclude syphilis microscopically, but the presence of many giant cells and the less definite perivascular sheathing might be considered in favor of another diagnosis.

Some General Considerations Regarding Clinically Similar Cases in Oceania and Elsewhere.

This case is, I believe, clinically indistinguishable from an affection of the naso-pharynx which I found very prevalent in Guam in 1902. I think it probable that it is met with more frequently there than elsewhere; and further, as I shall attempt to show, that it is sharply delimited upon neighboring islands. It is altogether likely that it is confined also to the tropics. Trusting to published cases alone, it seems to occur in tropical America (Brazil, Chili, etc.), in the South Seas and in Ceylon. Spain may have disseminated it; for example, quite the majority of the copper coins current in Guam are from Chili and Peru.

I saw about thirty cases of this disease while I was familiarizing myself with the conditions there in the capacity of health officer of Guam and senior surgeon of the U. S. Naval Station. The whole number of cases upon the island is estimated at from one hundred to five hundred, or from one to five per cent. of the entire native population. I studied none of them exclusively, owing, first, to the pressure of other duties and, afterwards, to illness for which I was sent away and I did not return. Clinically, however, I thought it competent to exclude yaws (whose existence there had not been emphasized), and syphilis—as it is known amongst the white races.

The usual history is that of a slight pharyngeal, tonsillar or nasal ulceration, which is more often noticeable from its discharge than from its painfulness, hemorrhage or other active symptoms. Constitutional involvement seems well-nigh absent throughout most cases. This ulceration spreads under superficial sloughs, which may disappear from time to time soon to re-form, the process extending by contiguity through the pharynx and nose. The palate and nasal septum soon disappear. The larynx is not often involved and usually escapes without notable damage. The eyes are often affected, being reached by extensions through the lachrymal ducts, and after the eyeballs and all of the secreting structures of the orbits are destroyed, the sockets may be filled up with granulation tissue and entirely cicatrized. I saw no cases of involvement of the middle ear, and the ulcerations of the posterior pharyngeal wall were slight and quite temporary. Through its course the tongue, cheeks and lips are very little involved, although the entire nose is often removed. Extensive destructive ulceration elsewhere is by no means common in these patients, they being, almost without exception, in the ordinary health of their class—certainly in no sense cachectic. The process may be rapid, although it seems to be apt to extend intermittently until after the destruction of the median and less vascular tissues of the naso-pharynx. Pain is not a prominent symptom and the disability depends, of course, upon the extent of the ulceration. Many distressing sequelae, such

as lachrymal fistulae, keratitis, etc., are produced; but the tendency is always towards self-limitation. I heard of no deaths ascribed to it.

Its association in families suggested contagiousness, but this is, at least, not of high degree. Virtually all ages were presented by the patients that I saw and no notable predominence in sex existed. The deformity is often of terrible extent, which acts in this well-favored simple race toward such concealment of the affliction as is possible. The disability involved phonation always, and usually, the senses of smell and taste—and perhaps vision.

These observations were, in effect, all confirmed by my successor in Guam—Surgeon James F. Leys,[3] U. S. Navy.

Daniels[4] noted its existence in Fiji; and it is figured—amongst yaws, syphilis and probably other affections—in natives of Ceylon.[5] Prof. Achille Breda,[6] of the Royal University of Padua, has made studies of allied affections presented by Italian emigrants returned to the neighborhood of Padua from Brazil. The illustrations of both of his papers show conditions somewhat different from the clinical appearances presented by Dr. Fordyce's patient and the histological findings are also somewhat dissimilar. Breda claims especially to have noted the indolence of the lesions of his cases and accentuates the priority of his record of the lesions of the mucosae. The inappropriateness of his name for the conditions that he studied—Brazilian framboesia—will be considered later; but his alternate designation of boubas, like parangi, forces confusion with syphilis, which, for my part, I am entirely willing to trust to Prof. Breda clinically to exclude. Sir William Kynsey[7] has pointed out the grouping together of most diverse conditions in The New Sydenham Society "Atlas of Clinical Medicine"; has explained that *parangi* is the same as the Ceylonese term *feringee,* which was the designation in Ceylon of the Portuguese, and has shown that this name among the natives of Ceylon includes syphilis and much besides. The colored plate in "Atlas" from his report on *parangi* in Ceylon is reproduced in Plate II, Fig. 1. It represents accurately the clinical picture often seen in Guam. Plate II, Fig. 2, shows a more advanced stage of the disease as seen in Guam.

It is perhaps exceptional for a visitor to Guam to escape seeing one of these pathetic creatures, notwithstanding the fact that such visitors are usually on shore there for a very short time. They are impelled almost unhesitatingly to pronounce these apparitions lepers; and, if these mistakes should be corrected, no effort will avail to upset the alternative conclusion that it is the result of syphilis. Even well-informed professional men jump thus at conclusions; hence, probably a part of the confusion with which the affection is surrounded. Undoubtedly the disease would have been more closely studied if such a number of horribly afflicted persons were to be seen in any of the more frequently visited tropical islands, such as Hawaii, Tahiti or the West Indies.

(See in this connection, the impressions of a young American woman who resided for a time in Guam, volunteering to teach there: "Johns Hopkins Nurses' Alumnae Magazine, May, 1905.)

Its absence from the other islands of the Ladrone (or Mariana) group —especially from Ponape is in striking contrast to its prevalence in Guam. I had this fact first from two Spanish Capuchin friars, both of whom I found to be good lay observers of medical matters. They came to Guam from Ponape in 1901. But I was most interested to find that Dr. R. Koch confirms[8] their observations, adding the evidence of Dr. Guschner, colonial surgeon at Ponape. Koch considered that numerous cases that were submitted to him and to the other members of the German Malaria Commission in the Caroline Islands as examples of lupus, of leprosy and syphilis, were yaws! Of course, it is inconceivable that such authorities as composed that commission should not recognize frank lupus, or leprosy, or syphilis (as syphilis is figured in temperate climates); and it is equally inconceivable that no typical cases of yaws should have been brought before them. Therefore, the inference is plain that the comprehensiveness of the conclusion expressed is due to slight acquaintance with yaws as well as to a well-nigh universal willingness to consider it a serious constitutional disorder of much the same formidableness as syphilis. Undoubtedly this commission saw cases such as have been described. Yet, as shown above, it recognized the absence of such cases from Ponape where yaws is endemic and almost as prevalent as anywhere else.

I have often shown well-marked cases of yaws to non-professional persons, in order to observe what their descriptions of the tubercles would be; and I have never yet observed anyone to omit to compare them to raspberries, whence, of course, *framboesia*. Charlouis[9] hoped vainly to lessen confusion in a locality where the pathological condition that occupies us seems to have been non-existent by replacing the opposite term *framboesia* with a histologically descriptive and a locative name. Jeanselme's personal investigations in Cochin China and Annam (reported at the meeting of the British Medical Association in July, 1905), and Cannac's[10] and Montel's[11] admirable and exact clinical contributions regarding yaws, appear to establish this disease as independent of syphilis. Doubtless, as A. Le Dentu says,[12] the alleged sequels of yaws and their relations to syphilis, may not be convincingly discussed until the whole question of tropical syphilis is elucidated; and I think that it may be admitted that this great field has been up to the present, if not entirely untilled, but little more than scratched. If, however, it be admissible to anticipate conclusions from facts already available regarding trypanosomiasis and from work now in progress relating to syphilis, it would appear that other diseases than syphilis and yaws will be

found responsible for various tropical ulcers which heretofore have been endlessly confused under geographical names.

As Leys suggests (*loc. cit.*), Guam affords an inviting field for original investigation in line with the case above reported. He observed more than forty cases similar to this one. They were all clinically disinguishable, as we have seen that the German Malaria Commission found, from leprosy, syphilis and tuberculosis; and his report includes exclusive reasoning that places it apart from yaws and establishes it as an affection *sui generis*.

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Plate I. Photograph of a negro, a native of Panama, showing ulceration involving the upper lip, the anterior nares and the septum. Also an infection of the right nasal duct. The tip of the nose is sunken from destruction of the anterior part of the septum.

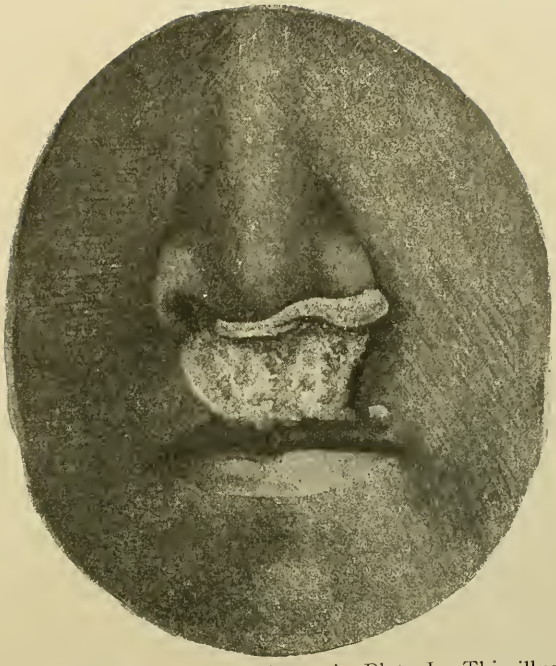

Plate II. Fig. 1. Condition like that shown in Plate I. This illustration is reproduced from The New Sydenham Society Atlas of Illustrations of Clinical Medicine. Surgery and Pathology. Fasciculus I and II of new series. Frambœsial Syphilis (Yaws and Parangi). Plate LXXXVIII.

Plate II. Fig. 2. Illustration taken in Guam, showing a more advanced stage of the same disease.

Plate III. Spencer ½-inch, Zeiss Compensation Ocular 4. Showing dense infiltration with numerous giant cells, extending deep into the derma.

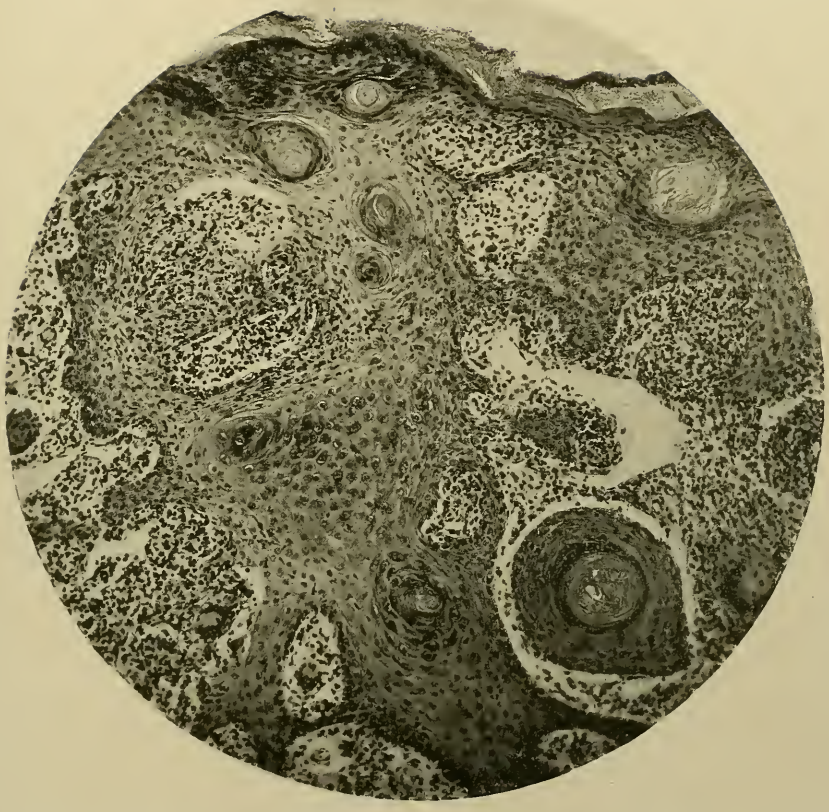

Plate IV. Spencer ½-inch, Zeiss Compensation Ocular 4. Hyperplasia and down-growth of the epidermis, with cellular infiltration of the corium.

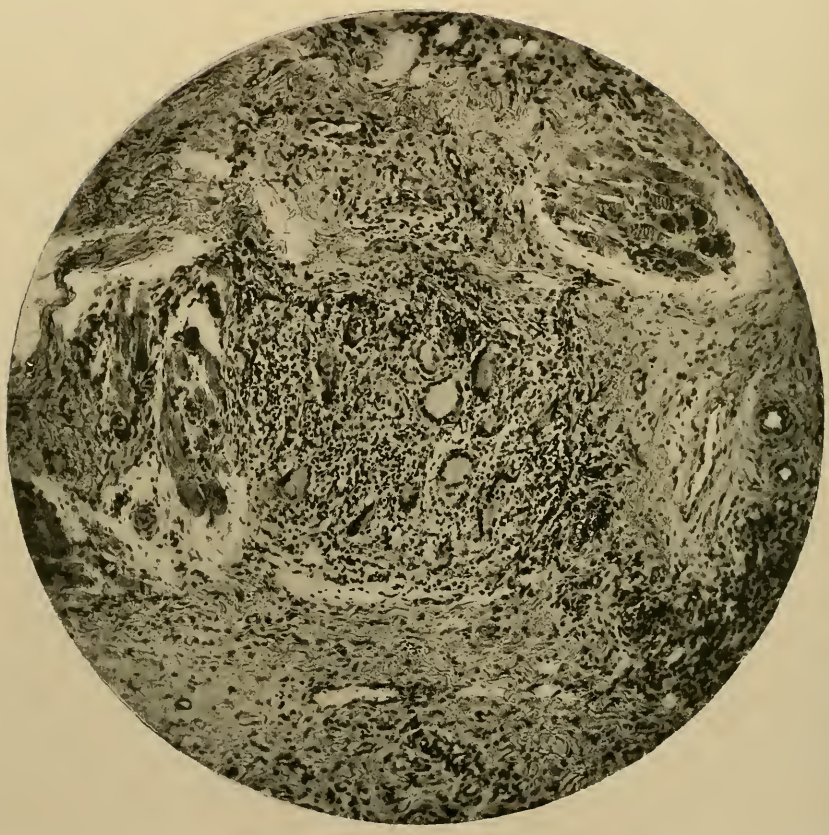

Plate V. Spencer ½-inch, Zeiss Compensation Ocular 4. A sharply circumscribed inflammatory focus with numerous giant cells, in the deeper layers of the corium.

Plate VI. Spencer ½-inch, Zeiss Compensation Ocular 4. Section of skin of inoculated guinea pig, showing infiltration made up chiefly of polynuclear leucocytes.

DERMATITIS VEGETANS IN ITS RELATION TO DERMATITIS HERPETIFORMIS.*

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The patient, whose skin affection forms the subject of this paper, has been under the observation of several of our colleagues and ourselves during the four years in which it has developed. On account of the unusual clinical picture several diagnoses have been made, such as dermatitis herpetiformis, pemphigus vegetans, blastomycosis, etc. A careful consideration of the evolution of the eruption, and a comparison with similar conditions reported, lead us to think it is worthy of record, even though its etiology may not be made entirely clear. In the localization of the lesions, as well as in most of the salient features of the disease, it resembled closely the group of cases reported by Hallopeau,[1] Hartzell,[2] Jamieson[3] and others. To Hallopeau is due the credit of first calling attention to the affection in a series of communications, but his opinion as to its nature has undergone some modification since his original article.[4] Whereas, in the beginning he looked upon it as a distinct entity, in later contributions he regarded it as the suppurative form of pemphigus vegetans.[5] Primarily, he reported the affection under the name of "Dermatite pustuleuse chronique en foyers à progression excentrique,"[6] but subsequently added the word "végétante" to the title, and in 1898 he included the five cases observed by him under the designation of "Pyodermite végétante,"[7] of which the following is a brief resume:

His patients were adults in whom the disease appeared without definite cause. The initial lesions were in the neighborhood of the genitals, lips, mouth or fingers, and consisted of vesico-pustules on an inflamed base. They increased rapidly in numbers, enlarged by peripheral extension, became elevated and the central part dried into a crust, which on removal exposed a dusky, papillary surface. Without symmetry, the scalp, neck, back and extremities were affected, and all transitions between the primary pustules and phlegmon, which was quite extensive in one case, existed. In the buccal cavity the efflorescences soon became papillary. The general condition remained good and the lesions disappeared under local antiseptics, the mouth and nose being more refractory to treatment. One of his patients died from an intercurrent erysipelas. Microscopic examination revealed an epidermic proliferation with vesicle formation, oedematous papillae and a cellular infiltration, of which 95 per cent. were eosinophiles. There was also a general eosinophilia of 16 per cent. From the pus he obtained bacilli which were not definitely classified. He concluded that Pyodermite végé-

* Reprinted from The Journal of Cutaneous Diseases, Vol. XXIV., 1906, page 543.

tante was a type which might develop primarily or, secondarily, in predisposed individuals in dermatitis herpetiformis or in pemphigus vegetans. If the latter, he did not consider that the complication established an identity with the two diseases mentioned, as the manner of its development and progression was by auto-inoculation, while in dermatitis herpetiformis and in pemphigus vegetans, the lesions appeared simultaneously under the influence probably of some internal toxin. Neither should the vegetations following these vesicles be confounded with pyodermitis, as the latter increased in numbers, might reach enormous proportions and have an asymmetrical distribution. Finally, in contradistinction to dermatitis herpetiformis and pemphigus vegetans, the disease had a favorable prognosis and yielded to local antiseptic treatment.

Hartzell[8] in 1901, under the title "Dermatitis Vegetans," reported a case in which the lesions occupied the groins, the inner side of the thighs and the legs. The two latter situations presented the objective features of a chronic eczema, of which intense itching was a marked symptom. According to the patient's statement, the eruption apparently began with pea-sized pustules on the buttocks and scrotum, which became confluent and crusted. Later it extended to the groin and thighs, but it seemed doubtful whether pustules were a part of the disease in the latter location. Vegetating lesions appeared in the groins. No pustules were ever observed by Hartzell, excepting an occasional miliary one upon the inguinal plaques. So far as could be learned, bullae had never been present, nor were the mucous membranes ever implicated. The patient succumbed to a catarrhal pneumonia three weeks after entering the hospital.

In 1902 Jamieson[9] described an eruption in a child which began at the side of the nail of the left middle finger. The nail was shed and the disease then involved the back of the hand and later the head and other places. The lesions corresponded in site and development to the description given by Hallopeau. Two noteworthy features were an unpleasant odor and the absence of itching.

Also worthy of special mention in a case of dermatitis herpetiformis with pustular and vegetating lesions presented by Wickham[10] before the French Society in 1891. His patient had been suffering from dermatitis herpetiformis for four years, with alternating periods of quiescence and pustular eruptions and had vegetations on his hand, perineal and scrotal regions. The lesions were deeply pigmented, papillomatous, surrounded by an epidermic collar and contained numerous abscesses. Extensive in places as the hand, where the entire surface was covered, they had the appearance of papules of variable dimensions elsewhere, as the anus. Their evolution was by eccentric progression, the point of departure being small pustules of auto-inoculable origin, which enlarged and became crusted in the centre.

When the crust fell off there was a vegetating pigmented surface, which by coalescence with similar plaques, produced large areas which were painful. The staphylococcus albus was isolated from the pus, and inoculations were negative. The presence in the same patient of lesions of a different aspect, Wickham contended, was to be explained not by a simple coincidence, but by the polymorphism of the eruption of dermatitis herpetiformis, and he was of the opinion that the various characteristics of his case approached those to which Hallopeau gave the name of "Dermatite pustuleuse chronique en foyers à progression excentrique." In the discussion Besnier, Vidal and Brocq agreed with the diagnosis.

Similarly one of us (Fordyce[11]), published a case of dermatitis herpetiformis with vegetations in a man of sixty-six, who had had many recurrences of an eruption of vesico-pustules during a period of forty years. The attacks would disappear and he would be free from the disease except for the pigmentation left by previous lesions. When he came under observation, the legs below the knees, the lower half of the thighs and the greater part of the forearms were the seats of a confluent eruption, made up of vesicles, pustules and papules, with an offensive sero-purulent discharge. It extended to the abdomen where there were a number of irregularly rounded plaques, the margins of which were crusted. Treatment had little effect and a short time after his admission to the hospital he developed constitutional symptoms, with another outbreak of the disease on the thighs, abdomen and back. The condition gradually improved and three weeks after he left the hospital. In a short time, however, he returned and was under treatment for another attack. He was lost sight of for about eight months, and when seen again he was cachectic looking; a papulo-vesicular and pustular eruption was present on both legs, the right leg was elephantiasic and papillary outgrowths covered the lower third of both legs and the dorsal surfaces of the feet. His appetite was fairly good and the patient said he had gained in strength. Histologically, the tissue resembled that which was obtained from the subject of this report. The identity of this case with the condition under consideration has been overlooked, probably because of the predominance of the herpetiform lesions over the pustular and vegetating ones.

The occurrence of vegetations in the course of skin affections has long been noted, and during the past ten years numerous contributions have been added to literature on this complication in impetigo contagiosa,[12] eczema,[13] seborrhoeic dermatitis,[14] inguinal tropical granuloma, pyogenic granuloma, iodide and bromide eruptions, dermatitis herpetiformis, pemphigus, syphilis, tuberculosis, blastomycosis, mycosis fungoides, impetigo herpetiformis, parasitic sycosis, chronic ulcerations, *i. e.*, ulcus cruris, epithelioma, etc.

The following history, from June 15, 1905, to November, 1905, was furnished by one of us (Gottheil) under whose care the patient first entered the City Hospital.

History—S. H., aged forty-two, clerk; no previous venereal disease. He had been in good health until the beginning of 1902, when an eruption consisting of "blisters" appeared on his scalp. A little later a similar eruption, but of larger and yellowish vesicles, came on the abdomen and then the right leg became involved. Since that time various parts of his body have been affected. The disease appeared on the forehead and spread thence over part of the face. It had been on the scalp, causing loss of hair; the penis and scrotum and the neighboring skin of the abdomen and thighs had been involved; so also had both legs from the knees down, and isolated smaller groups of lesions had appeared on various other parts of his body.

During the three and a half years that elapsed since the disease first attacked him, he had never been entirely free from it. It appeared usually in different areas; the affected surfaces took a long time to heal; and long before they were well, there would be a new outbreak. In addition to other treatment, he had been X-rayed over the lower abdomen, which he thinks benefited him, and he had a burn resultant therefrom still unhealed. The diagnosis at the Skin and Cancer Hospital at that time was, he says, pempigus vegetans.

Status praesens, June 15, 1905. Patient was in fairly good health, though feeble, and apparently much older than he claimed. Pulse, respiration, temperature and general functions normal; he was out of bed since the lesions of his eruption were all healed or healing. There was little or no hair over the front part of his scalp; and that area as well as the forehead and scalp showed very superficial cicatricial areas. These as well as other scars were, he claimed, the result of antecedent outbreaks. On the right side of his chin was an area in part cicatricial, and in part still eroded and crusted. On the neck, front and sides and over the sternum and upper half of the chest was a large, reddened and superficially cicatricial area, where the process had nearly ended, but which still showed a few ulcerated and crusted foci. The lower abdominal region, the sheath of the penis and the scrotum were the seat of a large healing ulceration. This was the X-rayed area which had very slowly been getting well during the past few months. The legs from the knees down and the upper surface of the feet and most of the toes were the seat of a hypertrophic, papillary dermatitis; the tissues were reddened, verrucous, not especially sensitive, with some foul secretion in the interstices of the masses and showed but little tendency to repair.

The patient was quite positive that all the lesions, with the exception of that on the lower abdomen and genitals, were the results of the vesicular eruption from which he had suffered.

June 20th to July 15th. A fresh attack of the eruption confirmed the patient's statements. Over several extensive areas a new crop of vesico-bullae appeared. In spite of the large territory involved, his temperature and functions were not disturbed; he remained in bed on account of the soreness and the dressings required. The largest area involved was that of the perineum, the adjacent thigh surfaces half way down to the knees, the scrotum and penis and the pubic region (Fig. 1). There were large areas of eruption also on the outer surfaces of the thighs (Fig. 2) and on the left leg, and there were a few isolated vesico-bullous lesions on other parts of the body. Some new lesions also appeared on the face and in the papillomatous tissue of the right leg. At all events this latter area, which had been getting drier and flattening down, became much worse, swollen and moist, and finally showed some eroded and secreting places.

The cruro-genital eruption had a very distinctly limited margin, especially on the thighs and buttocks. (Fig. 2.) This was also the case on the left leg; but on the outer surfaces of the thighs it was less sharply circumscribed and scattered lesions spread out far into the surrounding skin.

The individual lesions came out in irregular crops at intervals of a few days, so that by the middle of July they were present in various stages. (Fig. 4.) They began as closely aggregated minute vesicles, which in a few days became large, flat pustules, filled with a yellowish serum. Many of them were very superficial; adjacent ones would run together to form irregular bullous figures, and in some places circinate lesions were observed, a central vesicle or dried crust surrounded by a bullous ring, looking like a multiform erythema lesion. When the flaccid bullae ruptured, or when, after drying up, the scab was removed, a circular eroded area was left behind.

August 1, 1905. The outbreak of new vesico-bullae ceased and the slow process of repair began. The penile sheath and the scrotum were eroded over almost their entire surfaces, and so also were the upper and inner surfaces of the thighs. The confluent bullae made the entire surface look as if it had been subjected to a severe burn. The papillomatous condition of the left leg became more pronounced; the overgrowth was larger, redder and more tender. The left leg became the seat of a similar though less accentuated papillary growth.

October 1, 1905. Healing progressed steadily in all the lesions but very slowly. The course was exactly like that observed in burns of the second degree.

Early in November the patient had another attack; it was very much more limited than the first one and showed no features different from those already noted.

The treatment during the six months under observation was arsenic internally, tonics and stimulants when needed, the local outbreak being treated like a burn, with wet boric acid or liquor Burowii dressings.

When the patient was seen in March of this year (1906), (Fordyce's service), he was poorly nourished and anaemic, with scars on his face, scalp, neck, abdomen, back, genitals, and upper third of the thighs. A deep and irregular pigmentation extended from his chest to the umbilicus, below which the integument was mottled from scarring and isolated pigmented areas. He had erosions on his palate and lip, as well as partial oral atresia due to inflammatory infiltration, which had evidently extended to the subcutaneous muscular tissue. His legs were swollen, the right to twice the size of the left, making walking difficult, and the skin here presented a reddened, thickened, and exuberant appearance, with miliary abscesses between the papillary growths. (Fig. 5.) New lesions appeared about the periphery of the infiltrated areas in a manner similar to those in Hallopeau's and Jamieson's cases, as small pustules on a hyperaemic base. They soon ruptured, leaving erosions which subsequenty became the seat of warty outgrowths. The disease spread to the plantar surfaces of the feet, and its extension could be traced by auto-inoculation on surfaces blistered by strong ammonia to obtain cultures from the normal skin, where lesions appeared identical with those which developed spontaneously. During the four months he was under the care of the reporter there were no acute outbreaks like those met with in dermatitis herpetiformis, the only new lesions being those along the borders of pre-existing plaques.

The patient also had a bilateral conjunctivitis and a keratitis of his left eye, probably the result of auto-inoculation. His further physical examination revealed dullness over both apices of his lungs and fine sibilant rales. He had a cough, but no tubercle bacilli were found in the sputum. There was a slight systolic murmur and moderate arterial thickening. The liver was felt two inches below the free margin of the ribs. A mass of lymph nodes was present in the left triangle of Scarpa.

His temperature fluctuated between 98° and 99.6°, there being a rise of about half a degree during the afternoon. The pulse averaged about 90.

The blood examination showed haemoglobin 75 per cent.; red cells 4,750,000; white cells 10,000, of which there were polynuclears 63 per cent., lymphocytes 9 per cent., transitional mononuclears 10 per cent., eosinophiles 17 per cent. and myelocytes 1 per cent.

The urine was amber colored, had a specific gravity of 1024 and contained a faint trace of albumin, occasionally a cast, but no sugar. His nitrogen elimination was greatly reduced, the total for 24 hours, in 1216 cc. of urine, amounting to only 5.80 grams. An accurate determination of the nitrogen intake was not made, but it was far in excess of that excreted, as he

had an unnatural appetite, eating not only his own meals, but frequently those left by other patients, and in addition he sent out of the hospital for more food. Notwithstanding his overeating, he had steadily lost in weight, from 147 to 115 lbs. The decrease in the amount of nitrogen eliminated was interesting in view of the fact that similar observations had been made in cases of dermatitis herpetiformis (Crocker).

The erosions, fissures and ulcerations about the mouth and genitals (Fig. 6) were extremely resistant to treatment. In spite of the use of strong silver solutions, balsam of Peru or mercurial applications, they healed slowly. The elephantiasic thickening of the legs was treated by wet bichloride dressings, aluminum acetate solutions, ointments containing ammoniated mercury and various antiseptic powders. The upper half of the infiltrated area responded to this medication; the verrucous proliferations over quite an extensive territory disappeared, but over the lower half of the leg the vegetations still persisted. He is now under X-ray treatment.

Histology—Pieces of tissue excised at different times were fixed and hardened in Müller-Formol, alcohol, Zenker's fluid and formalin 10 per cent. and stained by the various nuclear, collagenous and bacterial methods.

All the sections showed epidermic hyperplasia, the degree varying with the duration of that particular lesion. The interpapillary pegs were everywhere much increased in size and penetrated the derma as styloid or club-shaped processes, some anastomosing and extending in a reticular fashion. The prickle layer was the seat of a moderate amount of oedema and mitotic figures were rather common. The granular layer in places was normal or missing, and in other sections it was thickened. The corneous layer likewise was not constant in its appearance, the picture being a hyperkeratosis in one part, a parakeratosis in another, or again, it was thinned or capped by a crust.

The most striking feature of the epidermis was the presence of vesicles which having developed in the deepest layers (Fig. 7), enlarged and extended to the surface. Their walls were formed by rete cells which were much elongated and narrowed through three or four rows, suggesting stretching. Their contents were composed almost entirely of eosinophiles. (Fig. 8.) Throughout the Malpighian layer migrated cells, isolated or in small groups, the initial point of a vesicle, were encountered. They, too, were largely eosinophiles, although there was an occasional polynuclear neutrophile.

In the corium the papillae were correspondingly hypertrophied. They were oedematous and evidence of more or less oedema was seen in the subjacent area. Apparently perivascular in the beginning, the infiltration had a more extensive distribution in older lesions, and while dense and diffuse in the upper portions of the cutis, it was rather closely confined to the vessels

below. Here again the eosinophile was the preponderating element and but few neutrophiles and lymphocytes were present. Mast cells were very abundant in some sections and plasma cells were numerous about some of the deeper vessels. Although so thickly crowded in the pars reticularis, individual eosinophiles were scattered in the depths of the cutis and also appeared in some of the vessels. There was an active proliferation of fibroblasts. In addition there were a peri- and an endarteritis, the lumina of many of the vessels being distended, others again, narrowed or occluded. The lymph spaces were dilated.

In some regions the elastic tissue had entirely disappeared from the upper two-thirds of the corium. Approaching the deeper layers fragments or granular bits were interspersed with normal fibres. In sections in which vesicles were absent, the elastic tissue showed no change except a slight amount of granulation in the pars papillaris.

Microscopically, the appendages were normal excepting for some mucoid degeneration about the coil glands.

Careful examination for organisms revealed nothing except numerous Gram positive cocci and a very few Gram positive bacilli in the superficial layers of the horny cells and in the crusts.

Smears made from turbid vesicles from various parts of the body, from the eye and from erosions about the mouth, showed Gram positive cocci and occasionally a small Gram positive bacillus, with slighty fusiform ends. The latter failed to grow on any of the media.

The bacteriological examination of material from the sites enumerated resulted in only a growth of streptococci and staphylococcus aureus and albus, sometimes pure, but more often mixed. Aerobic and anaerobic cultures were made, Loeffler's blood serum, plain, glucose and glycerine agar, plain and ascitic bouillon, as well as the various sugar media, having been tried. From the normal skin cultures on different media produced only the aureus. The search for blastomycetes in smears and tissue was negative.

Cultures from the blood remained sterile.

The significance of eosinophilia in dermatitis herpetiformis is not of so much importance as at one time thought; still a large percentage of these cells in the fluid contents of vesicles, as well as in the tissues of the derma, would seem to point to a special irritant which had called them forth. In addition to their presence in this disease, they have been found in pemphigus, eczema, scleroderma, psoriasis, pellagra, lupus, if widespread, urticaria, leprosy, bromide and iodide eruptions, parasitic diseases, gonorrhoea, tumors, etc. Ehrlich is quoted by Ewing[15] as believing that substances which attract eosinophile cells may be derived from the destruction of epithelial cells, as seen in the local eosinophilia about the ulcers of lupus, after the injection of tuberculin. Furthermore, Ewing[16] explains the phenomena connected

with general and local eosinophilia by the same chemotactic principles that are known to control neutrophile cells. It would appear that inflammatory products attract eosinophile cells at one stage and neutrophile cells at another and more acute stage.

In its clinical and histological features, the case which we have cited is practically identical with Hallopeau's, Jamieson's, Hartzell's, and others previously reported. Whether it is to be considered an independent affection, a variety of pemphigus vegetans, a complication of dermatitis herpetiformis, the result of pyogenic infection, either directly or from the toxic products of the organisms, are questions more easily asked than answered. In the last few years more attention has been paid to this complication in dermatology, notably in the disease in question, as well as in those due to inoculation of pus organisms in seborrhoeic eczema (*e. g.*, the cases of Wende and De-Groat[17] and of Perrin[18]).

The presence of vegetations in so many affections in which pyogenic organisms are the etiological factor, suggests that they or their toxic products are concerned in the production of the condition. This would account for their occasional occurrence in eczema, impetiginous inflammations and in all skin diseases where secondary staphylogenic or streptogenic infection is met with. The older writers laid much stress on irritating discharges and secretions as significant in producing this form of epidermic hyperplasia, as in pointed condylomata about the genitals from gonorrhoea and allied conditions about the lower extremities in chronic eczema and in chronic ulceration. It would seem that in some instances where vegetations are a part of the clinical picture, that a chemical irritant might be provocative, as in the papillomatous lesions met with in certain iodide and bromide eruptions. If this could be demonstrated, it would not be necessary in every case to invoke a microbic agent to explain epithelial hyperplasia. It cannot be denied, however, that in iodide and bromide eruptions, a secondary pyogenic infection is possible if not probable. The development of vegetations in such a variety of diseases leads us to believe that it should be looked upon rather as a complication than as an essential feature.

In our case, although the grouped vesicular eruption which appeared about the scalp and face, and the lesions of the thighs and lower extremities suggested dermatitis herpetiformis to one of us later observations rather negatived such a diagnosis, for the reason that there were no acute generalized outbreaks and little or no pruritus. Then, too, the peripheral extension of the affection could be distinctly traced by auto-inoculation and the conjunctivitis and keratitis might be explained in the same manner. It cannot be denied, however, that the early lesions decidedly favored the diagnosis of dermatitis herpetiformis, and that the histological examination and the presence of eosinophile cells in the blood and vesicles strengthened this view.

On the other hand, the pronounced scarring about the abdomen and genitals (probably in part due to the X-ray employed), the face and scalp from the deep-seated persistent lesions could not be accounted for on this supposition.

It would seem that there is a group of conditions which have been described under the names of impetigo herpetiformis, dermatitis herpetiformis, pemphigus vegetans and *pyodermite végétante* which have many clinical and microscopical points in common. That transition types are met with which present the features of several of these maladies is not unlikely. Pemphigus vegetans, however, is so constant in the localization of its lesions, its severe constitutional symptoms and its almost invariably fatal course that we are scarcely justified in placing it in the same category with vegetating dermatitis.

Conclusions—The case herein reported is the counterpart of Pyodermite Végétante of Hallopeau and the Dermatitis Vegetans of Jamieson and Hartzell. It presents certain analogies to dermatitis herpetiformis in its histology and its blood examination (eosinophilia). In its predilection sites, the mouth, genitals and lower extremities, it is very like pemphigus vegetans, but dermatitis vegetans in its relatively benign course is a striking contrast to the latter. If we assume that the disease in its inception was a dermatitis herpetiformis, the late persistent and vegetating lesions could be explained by reason of a secondary pyogenic infection. We are rather inclined to agree with the earlier opinion of Hallopeau, with which Hartzell and Jamieson concur, that the affection presents closer affinities to dermatitis herpetiformis than to pemphigus vegetans.

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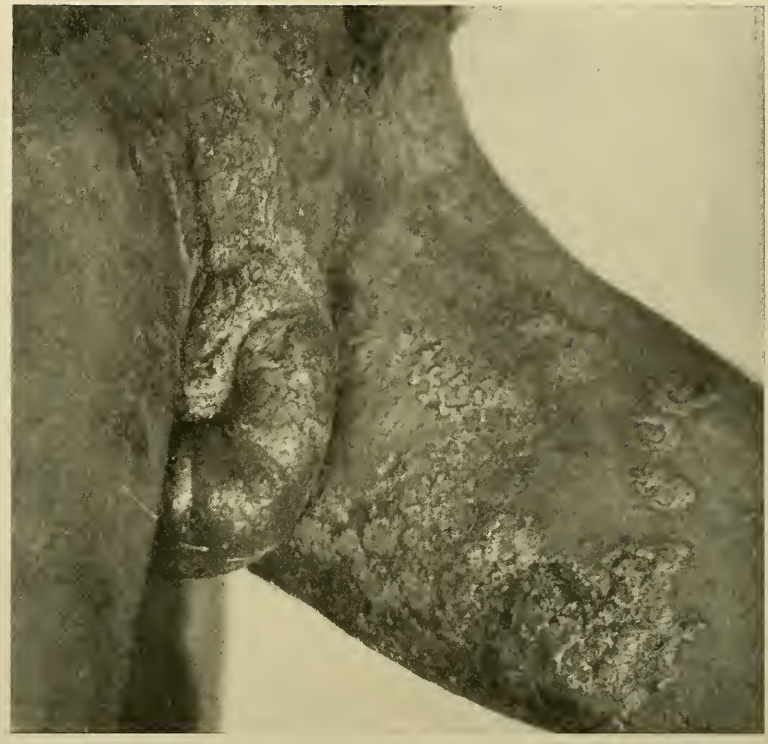

Fig. I. Showing vesico-bullous eruption about the genitals and inner surfaces of the left thigh.

Fig. 2. Showing presence of eruption on buttocks and over genito-crural region. Margins sharply limited.

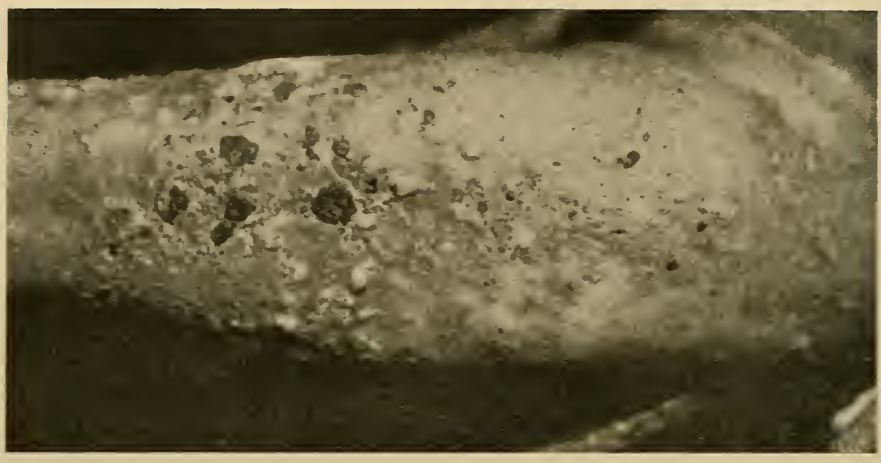

Fig. 3. Scattered vesico-bullous lesions over the thigh and leg.

Fig. 4. Showing scarring of abdomen, grouped vesico-bullous eruption on genitals and thighs, with similar lesions on legs before the papillomatous development. Figures 1, 2, 3 and 4 from photographs taken in July, 1905.

Fig. 5. Photograph taken in March, 1906. The skin over the legs is thickened, reddened, and the seat of a papillary growth interspersed with numerous miliary abscesses. The right leg presents a pseudo-elephantiasic appearance.

Fig. 6. Showing erosions, fissures and ulcerations about the mouth and chin. The superficial reticulated scarring about the scalp and right side of the face is not well shown in the photograph. The skin over the neck and upper part of the chest is the site of superficial scarring and pigment changes.

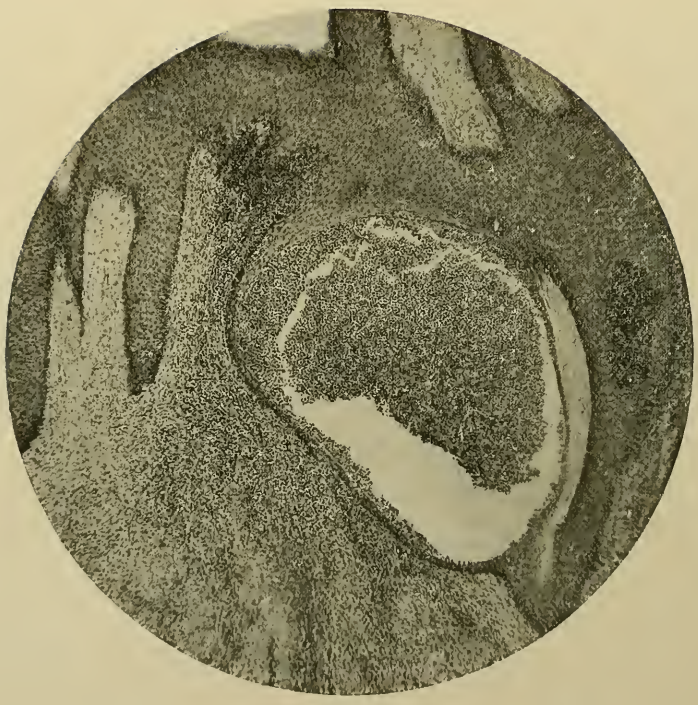

Fig. 7. Showing acanthosis, deep-seated vesicle in the epidermis and cellular infiltration in the derma.

Fig. 8. More highly magnified view of an epidermic vesicle, contents chiefly eosinophiles.

VII.—Ophthalmological Division

A CASE OF PANOPHTHALMITIS IN TYPHOID FEVER.*

By W. Whitehead Gilfillan, M. D.,

Attending Ophthalmologist.

Panophthalmitis is an inflammation of all the tissues of the eyeball. It may be caused by infection from without due to penetrating injuries or unsuccessful operations. This is by far the most frequent cause. It may also be produced by infection from within through embolism, septic substances from a focus of suppuration getting into the circulation and becoming arrested in the vessels of the choroid and other diseases. The case reported here is of the latter class.

Panophthalmitis is one of the most painful of eye diseases. Fortunately we do not observe it frequently. In the treatment of 20,000 cases in our eye hospitals about six or eight cases will be seen. Nearly all of these being due to the causes first mentioned. It is well we meet it no more frequently, as it is very destructive to the eye. The sight not only is lost, but also the shape, which results in a shrunken eyeball (Phthisis bulbi). The inflammation may extend to the meninges and brain and produce death. This is a rare occurrence, however.

When the inflammation gets well established there is nothing that can be done to save the sight. Panophthalmitis complicating typhoid fever is a very rare occurrence. Less than a dozen cases have been reported.

The patient, a young man thirty-one years of age, entered the medical service of Dr. Van Horn Norrie at the City Hospital, Blackwell's Island, August 25, 1902. I am indebted to Dr. Norrie for his kindness in letting me report this case, and also to his house physician, Dr. H. M. Little, in giving me the full history and temperature chart.

The patient had complained of loss of appetite, pain in the abdomen, vomiting and diarrhea for the two weeks preceding his admission to the hospital. These symptoms continued. The temperature was 103° F., pulse 100 and respiration 28. Examination of the urine showed it to be alkaline. Specific gravity 1.015, and a small amount of albumin. The microscope revealed a few hyaline and granular casts. He was fairly well nourished but his cheeks were flushed and the expression apathetic as well as the mucous membranes being pale. The tongue was coated.

The left eye, the one which was subsequently affected by panophthalmitis, had no sight. This was due to an injury received some seventeen

*Read before the Society of the Alumni of the City (Charity) Hospital, March 11, 1903.

years before resulting in an occlusion of the pupil. The diagnosis of typhoid was made.

August 27, two days after admission, a test was made of the blood. It showed Widal's reaction for typhoid in dilution of 1 to 20; leucocyte count 3,000.

August 29. Patient was very delirious during the past night and quite irrational this morning. Leucocyte count to-day, 2,600.

August 31. Abdomen distended and tympanitic.

September 1. Pulse slightly irregular and weak.

September 2. Tympanitis has disappeared. Pulse much improved.

September 5. This is the twenty-sixth day of the disease. To-day the outer part of the conjunctiva of the left eye is somewhat swollen and edematous; no discharge; a saturated solution of boric acid ordered every four hours, and ice applications every other hour to the eye. At five o'clock in the afternoon the eye was much worse. Lids, the upper one especially, very much swollen and edematous, with considerable redness of the skin. The conjunctiva is intensely reddened and likewise very edematous. The cornea dull with a diffused cloudiness. The eyeball is markedly protruded (exophthalmus); no discharge.

September 6. Eye still further protruded and the tension is very much increased.

September 9. The condition of the eye is the same except for a slight mucopurulent discharge. I was called in to-day to see the case and the diagnosis of panophthalmitis was made; patient very weak and unconscious. Treatment changed to hot applications, for fifteen minutes every half hour, to the eye.

September 11. Eye somewhat improved. Discharge increased and more purulent in character. A small opening in the anterior sclera allows the discharge to escape.

September 13. Eye much better; swelling of the lids much less; discharge continues free.

September 16. Swelling of the lids entirely disappeared; eyeball beginning to contract; discharge less.

September 29. Patient sitting up; discharge has stopped.

October 3. Eye much shrunken; treatment discontinued. Eye is now a phthisical one.

In regard to the typhoid condition, the patient ran a regular fever course as the temperature chart will show. The panophthalmitis ran a rapid course. The duration generally being about six weeks to two months, whereas in this case it took only a month.

Fortunately for the patient the disease affected the eye which already was blind, otherwise he would have been in a sad plight. I do not think the injury seventeen years before had left any condition which would predispose to this disease.

It will be noticed that this complication did not begin until the twenty-sixth day of the fever. By this time the height of the fever had been passed.

The patient was treated by the cold bath method for his fever and was discharged cured from the hospital the last of October.

44 West Fiftieth Street.

A CASE OF SARCOMA OF THE CORNEAL LIMBUS.

A. N. Strouse, M. D.,

Attending Ophthalmologist.

More than ten years ago I presented to the section on Ophthalmology of the New York Academy of Medicine, a case of sarcoma of the corneal limbus in which the growth had been successfully removed. In my remarks at that time I referred to the comparative infrequency of these tumors, and this fact prompts me to place on record a second case observed in December, 1906, at the City Hospital.

The patient, S. R., was admitted to the hospital with the diagnosis of Rupture of the Sclera with Prolapse of the Choroid.

The history given was as follows: Two years ago the right eye was injured in a street car accident. The eye was not treated and vision was not noticeably affected. About one week prior to admission to the hospital, the patient fell and again injured his right eye.

On October 26, 1906, the condition was described as follows on the history slip: There is a wound of the sclera with a protrusion of part of the choroid.

On December 1, 1906, when I first saw the patient, I found at the outer sclero-corneal margin a dark bluish-black prominence about the size of a large pea, irregularly round in shape, constricted at its base, not painful nor very sensitive to the touch, but interfering somewhat with the closure of the eyelids.

In view of the history and the previous diagnosis, I was inclined to watch the progress of the case before determining upon treatment. After observing the condition for three weeks and finding that the tumor was growing, but that otherwise conditions had not changed, I decided to remove it by ligation.

The patient was taken to the operating room and after thoroughly anaesthetizing the eye with cocain solution, a silk ligature was placed about

the base of the tumor and slowly and gradually tightened, my object being to excise the mass above it, leaving the ligature in place. However as the ligature was tightened it readily cut through, owing to the friability of the tumor. Hemorrhage was quite profuse and as the mass fell away exposing its site of attachment, it was seen to spring from the corneal limbus and that there was no evidence of rupture of the sclera. This at once convinced me that I had to deal with some form of neoplasm, most likely a sarcoma of the limbus.

The base was now thoroughly curetted and cauterized with the actual cautery, after which a moist bandage was applied over the eye. The patient suffered very little inconvenience and in the course of a week the slough had fallen off leaving a clean granulating surface. At the outer edge of the wound there was a small dark patch which I decided to remove and cauterize, regarding it as a small remnant of the growth which I had overlooked at the first operation.

The man made an uneventful recovery, and was later employed in the hospital, where he remained for several months, during which time there was no return of the growth, and I have since learned that six months later there had apparently been no recurrence.

The pathological examination was kindly made for me by Dr. H. Oertel, who furnished the following report:

Section of the specimen showed a diffuse growth, made up almost entirely of large and small cells, for the most part round, with large, richly chromatic nuclei and a small amount of interstitial stroma. A number of these cells contained characteristic pigment. A definite structure of the tissue or even arrangement of these cells, could not be made out, nor was any normal structure to be seen in this tissue.

The diagnosis of melanotic sarcoma was, therefore, made.

In my previous case I removed the growth by curettage and the cautery, and while recurrences took place these were easily controlled in the same manner.

I am aware that some ophthalmic surgeons would regard such a procedure as too conservative and would resort at once to enucleation. From my own observations and a careful study of the cases reported I am convinced such radical treatment is never required.

Examinations of eyes enucleated for sarcoma of the limbus have always shown that there have been no invasion of the deeper structures, no glandular involvement and no metastatic extension. This in itself indicates that the process is truly a local one and should be treated by local measures, and that recurrences should be dealt with in a like manner.

VIII.—Pathological Division

SYNANCHE* CONTAGIOSA (DIPHTHERIE) IN ERYSIPELAS, AND ITS RELATION TO CROUPOUS AND DIPHTHERITIC INFLAMMATION IN GENERAL.†

By Horst Oertel,

Director of The Russell Sage Institute of Pathology.

Before entering upon the report of an unusual complication of erysipelas by an additional infection of so-called diphtherie bacilli in the throat, I feel called upon to qualify the term synanche contagiosa. In doing so I enter upon a much confused field of pathology which demands importance, not only for academic and scientific discussion, but on account of its great practical bearing. Here, as in many other fields, popular ideas have become so deeply implanted that strict scientific classification has been greatly handicapped, and correct conceptions made difficult.

In 1826 Bretonneau presented in a fundamental work a description of certain characteristic inflammatory changes of the mucous membranes of the upper air passages which were associated with the formation of a pseudo membrane. He coined for this the name diphtherite (from διφθέρα skin, covering), and translated it "inflammation pelliculaire." He further stated that he was able to produce such "inflammation pelliculaire" experimentally by applying cantharidin to the upper air passages. It, therefore, follows that this term diphtherite was formed entirely on an anatomical conception. Diphtherite was taken over by the Germans who translated it diphtheritis, a most unfortunate term, because following the usual method of expressing inflammations of a part by the addition of itis, diphtheritis really means inflammation of the pseudo membrane. This, of course, is absurd; such an idea was never entertained. Bretonneau himself later changed the term to diphtherie, while the English took the word διφθέρα itself which was popularly and wrongly changed to diphtheria. In conformity with this anatomical conception introduced by Bretonneau, the terms diphtherie and diphtheritic were taken over by the pathological anatomists to signify all those inflammatory conditions which are associated with the formation of a pseudo membrane. It was mainly due to the efforts of Cohnheim and Weigert that a more exact knowledge of the pathogenesis of the pseudo membrane was established. Two features distinguish it: necrosis and fibrinous exudation. But inasmuch as these appear in more or less modified form, two subdivisions have been made; one in which the pseudo membrane consists of necrotic surface epithelium to which may be added coagu-

* In the spelling of this word I have followed Orth and Senator.

† From The Russell Sage Institute of Pathology, New York City Hospital.

lated fibrinous masses, remains superficial and does not extend to the mucus membrane; it can be stripped off without difficulty. This is croupous inflammation. In the other, it extends deep into the mucus membrane, involves this in the necrosis and with the fibrinous exudate and hemorrhagic extravasation forms a very adherent membrane, which can only be removed with great difficulty, and leaves a marked loss of substance, with a deep hemorrhagic ulcer. This is true diptheritic inflammation. Cohnheim and Weigert showed conclusively that the essential feature of both consisted in a primary necrosis of the fixed tissue. No diphtheritic or croupous inflammation is possible without death of all layers of the epithelium; then only the exudate assumes its distinctly fibrinous character associated with that peculiar form of necrosis which Weigert so appropriately termed coagulation necrosis. Living epithelium prevents the coagulation of the exudate. The manner in which the epithelium is destroyed is irrelevant, nor is its future fate of any consequence; its destruction is not the cause of the coagulation, but makes it possible. The same holds true in regard to the diphtheritic inflammations involving the deeper layers of mucus membrane. Without preceding necrosis of the fixed tissue, no fibrinous exudation, no coagulation necrosis and, consequently no diphtheritic inflammation. Subsequent investigations have mainly confirmed these views, and in the recent very detailed report of Councilmen, Mallory and Pearce, a very similar stand as to the course of events in the formation of diphtheritic inflammation is taken. But we have learned—and I shall refer to this later—that the stage of fibrinous exudation is not always reached, for the whole process may remain at the point of necrosis of the epithelium, or fixed tissue. This presents itself in the form of a granular detritus lying on the surface, or it may undergo hyaline degeneration. Then the mucus membrane appears glassy hyaline, a condition frequently seen in the throat in synanche.* At any rate, following the very exact and thorough investigations of Cohnheim and Weigert, the term diphtheritic was applied to inflammations of mucus membranes associated with the formation of a pseudo membrane. In this way it became quite proper to speak, for instance, of a diphtheritic colitis and enteritis in dysentery as well as of a diphtheritic gastritis and a pharangitis of varying etiology. Pathological anatomists have retained these terms to the present day.

But, while Bretonneau had selected the name of diphtherite on an anatomical conception, he had really introduced it to characterize a more

*It can easily be seen that the distinction between croupous and diphtheritic inflammations is mainly one of degree and, inasmuch as no special pathological conception is associated with the term croupous, Orth recommends to discard the term croupous altogether from the anatomical terminology and speaks of angina fibrinosa. On account of the confusion arising from the words diphtheria and diphtheritic he also discarded them and speaks of superficial and deep angina fibrinosa.

or less well defined clinical entity, which was later found by Trousseau to be contagious. Bretonneau had spoken of it as diphtherite undoubtedly because, in his opinion, the pseudo membrane was the distinctive feature of the disease; but subsequent clinical experience showed that the pseudo membrane could not be regarded as the only and characteristic feature of it. Indeed, long before any definite knowledge of any specific etiology of the disease had been obtained, it was well known that the pseudo membrane was not always present, and scientific clinicians—and especially the pathological anatomists—came into difficulty, for they were obliged to describe cases as diphtheritic and non-diphtheritic, that is, diphtherie with and without pseudo membrane. This, of course, was absurd because diphthera means pseudo membrane. A further discussion arose as to what constituted the distinctive character of the diphtheritic lesion. Some allowed it only then when all the anatomical data of an adhering membrane were present; others paid more attention to the location; the third group used the term etiologically to designate a definite clinical entity, and finally the pathological anatomists used it as a descriptive term without regard to location or etiological feature. It can be plainly seen that even before the days of etiological classification of diseases by specific bacteria, confusion as to the characteristic feature of this disease had become great. As early as 1874 an attempt was made by Senator to create a better understanding and to clear the confusion of clinical, etiological and anatomical ideas by reviving the time-honored term synanche to which he added contagiosa, and applied it to that clinical symptom complex known as diphtherite, diphthereitis, diphtheria, or diphtherie.

The term synanche, *συνάγχη* while it may appear new to some, is really a much older word than the term introduced by Bretonneau. From the investigations of Virchow it appears that Hyppocrates used this term for inflammatory lesions of the pharynx and trachea which were associated with respiratory difficulties. The term is not synonymous, however, with the so-called angina tonsillaris for which the expression *ἀντιάς* was used. Synanche or cynanche as the term signifies, was derived from *κυων* dog. Caelius Aurelianus explains its use in the way that dogs and wolves are frequently suffering from throat diseases, and that patients ill with throat conditions produce sounds resembling those of the animals. On the other hand, Aretaeus derives the word synanche from a disease of dogs, or because they frequently protrude their tongues from the mouth and allow the saliva to drop from it. Virchow thinks that the latter suggested the use of the term, because in the works of Hyppocrates considerable attention is paid to the flow of saliva in these conditions, and that Anche, *ἄγχειν* was probably derived from the fact that in severe cases the expression of the face resembles that of strangulated persons. It appears to me not unlikely that the word synanche (which really means dog collar), owes its introduction

into medical terminology to the fact that patients suffering from the disease produce a choking cough or sound which resembles very much that made by a dog when he is strongly pulled by the collar. At any rate, the term synanche came into general use and was used by Alexander von Tralles, Galenus and Celsus, and while occasional subdivisions were made, it signified prominently a severe inflammatory lesion of the upper air passages associated with considerable constitutional disturbance. The attempt of Senator was, then, not to introduce a new term, but to revive a much more general expression for a clinical entity which had been overshadowed by the anatomical conception of Bretonneau.

It is likely that on account of these efforts on behalf of some clinicians and pathologists (notably among the latter is Orth), a successful classification would have been accomplished if the results of bacteriological investigation had not interposed further obstacles. For, inasmuch as in synanche contagiosa the presence of a specific micro-organism was demonstrated by Loeffler, the fundamental mistake was now made to name this micro-organism diphtherie bacillus, which, correctly interpreted, means that it is the etiological factor of diphtheritic inflammations.* This, however, was a great mistake in more than one way, for the reason that not all diphtheritic inflammations of the mucous membranes, even in the throat and mouth, are caused by the so-called diphtherie bacillus (I may refer only to scarlet fever), and, on the other hand, the disease caused by this diphtherie bacillus may occur without the production of the diphthera. Evident contradictions! It introduced a new difficulty to both clinicians and anatomists, for no one knew any more (and does not even now), how to properly apply the terms diphtheritic and diphtheria. In discussions of modern clinicians and pathological anatomists, one cannot help being surprised how the terms croupous, diphtheritic, diphtheroid and pseudo membranous are juggled, and probably few have the same conception as to the nature of the process. Unfortunately all these terms mean the same thing, which does not aid the effort of those who wish to correct confounding etiological and descriptive classification. Badly chosen is the name pseudo-diphtheria for cases with a false membrane, but absence of Loeffler's bacilli. It means a disease characterized by a pseudo membrane, which is quite absurd, and further causes confusion with *pseudo*-pseudo membrane, which is quite absurd, and further causes confusion with the equally badly chosen term pseudo-diphtheritic bacillus. This has absolutely nothing to do with it.

* When I ask in my hospital examinations: What is a diphtheritic inflammation? the answer is usually: an inflammation produced by the diphtherie bacillus. It illustrates that the student of the present generation has evidently lost all idea that there are other diphtheric inflammations than those produced by Loeffler's bacillus. Some even believe that the diphtheritic inflammations of the intestines (as we find in dysentery) must be the result of infection by this bacillus. This is, of course, absolutely wrong, but it is really not the student's fault that such impressions are obtained.

It would be unfortunate if it were thought here that I raise merely academic questions, or a battle of words. This is by no means my intention. The chief danger of this confusion is that it interferes to a very great extent with uniformity of our anatomical conceptions, which must always remain the proper basis for scientific discussions of diseased conditions, for progress in pathology depends essentially upon clear anatomical conceptions. As this is not confined to the one instance under consideration, I may perhaps be pardoned for wandering away from our present topic and mention another example of disregard to correct anatomical conceptions and the harm done by it. The word endarteritis, which really means an inflammatory condition of the arteries of some kind or another, and affecting the intima, is now used by many as a collective name to designate all sorts of various arterial changes. As a consequence every time anyone speaks of endarteritis we are obliged to ask for a full description of the lesion before we can even understand what is meant by what the speaker choses to term endarteritis.* Another instance: the terms tubercle and tuberculous are used by many in an extremely wide sense. If anyone speaks to-day of tuberculous pneumonia there is no certainty what condition of the lungs exists. Correctly interpreted, it should be one characterized by the formation of tubercles, but more often is meant a cheesy pneumonia. This term, although old, is much to be preferred, for in the first place this lesion is especially characterized by the absence of tubercles, and represents a diffuse exudative process which becomes cheesy; and in the second place we can combine with this term a particular anatomical picture, that is a definite occurrence. But, even worse, I have often heard tuberculous pneumonia used in an even wider sense to indicate a peribronchitis, a perivasculitis, a broncho-pneumonia, or a lymphangitis or a perilymphangitis of the lungs by extension from the pleura and lymphoid glands of tuberculous origin or a vascular miliary tuberculosis. Tuberculous pneumonia then includes a great many lesions which represent distinctly different modes of entrance, pathogenesis and histogenesis. But the knowledge of these anatomical factors is undoubtedly of at least equal importance as that of the etiological factor, for the questions of entrance, of localization and generalization of an infective agent are purely anatomical and histogenetic problems. However, these are getting more and more to be disregarded in the terminology. In other words, we are at present unable to form any idea of the character of lesions by many of the terms used. This is not only a time-consuming and unnecessary matter but, worse, it is confusing and quite detrimental to scientific progress.

If we continue to complicate our nomenclature by mixing etiological, clinical and anatomical conceptions in such an indiscriminate manner as has been done during the past decade, we are in great danger of returning to

*This happened only recently at a meeting of the New York Pathological Society.

the primitive position of the times before the establishment of cellular pathology. Our knowldge, instead of more profound, will become superficial again and we will lose that sound conception of the disease itself and its history within the organism which has been so carefully developed by Morgagni, Rokitansky, Virchow and their followers. Without going any further I believe I have offered a foundation for the opinion that terms designated to cover clinical entities shall be more properly based on symptoms than on etiological or descriptive anatomical terms, for the reason that the last two are more complicated and constantly changed by the progress of research.

Now to return to our present topic. If we consider all these factors, I think we should mainly agree with Orth, who follows Senator, and whose views I have adopted, with some modifications. There exists a pseudo membranous angina which may be croupous or diphtheritic in an anatomical sense. A variety of factors may cause this; one of them is the infectious material of synanche contagiosa. This latter is a clinical entity, which is regarded as being excited by a specific micro-organism, the clinical manifestations of which, however, depend upon the presence of other factors as well. Synanche contagiosa may be associated with a catarrhal condition of the throat only, but it disposes a tendency to superficial and deeper necrosis of the fixed tissue which may or may not become associated with the products of fibrinous exudation.

Recently I have had an opportunity to study a case of severe erysipelas which was terminated by an additional synanche infection. Besides the general interest which the case has from a standpoint of mixed infection and the rarity of the lesion, it proved instructive as it allowed an insight into the changes which clinically presented themselves as oedematous reddening of the throat only, without the formation of a definite membrane. We are not often able to study such cases anatomically and histologically, because they either tend to recovery, or they advance to more destructive lesions which obscure the initial change. In the case under consideration, it is quite probable that the opportunity for this study would not have been obtained (at least not so early), if the previous streptococcus septicaemia had not been so severe that the additional synanche infection led to speedy exitus. It concerned a young Russian, about twenty-five years of age, who was admitted to the City Hospital on November 29, apparently convalescing from an erysipelas of the face indicated by a pretty general desquamation of the skin of the face. There was, in addition to that, at the jaw on the right side a fluctuating small tumor mass, which had been incised before admission and represented an infected purulent gland. Through the sinus of the old incision there was some purulent discharge, otherwise the patient presented no

characteristic features. He had a normal temperature which was taken during the first five days; and then, on account of the progressing good recovery, was discontinued. During the next week the patient refused further incision of the gland so as to allow better drainage. On the 7th of December his erysipelas of the face recurred; it involved both sides and the nose, and lasted for about a week, after which it somewhat subsided. During that time he ran a slightly irregular temperature which, in the beginning, was somewhat high (to 103° F.), and then, at the end of a week, moderated again. But on the 14th of December his erysipelas began to spread to the neck and the back down as far as the sacrum; it became very extensive and also involved the left arm. His temperature now began to run a pretty typical septic course, and continued so until there was a remission of all the symptoms, beginning about the 24th of December. Temperature, pulse and erysipelatous swelling and reddening subsided gradually, and from the 27th of December he practically had a normal temperature; his pulse had gone down to 80, and respirations were about 22-24. On the 31st of December, the patient for the first time complained of difficulty in swallowing; he felt nauseated and he did not take nourishment well, apparently on account of his difficulty in swallowing; he did not sleep well during the night and coughed a great deal. On the 1st of January, 1908, he complained considerably more about his throat, and on examination the tongue appeared swollen and oedematous, rather thick; it was apparently covered by some blood crust and the same condition appeared at the posterior roof of the hard palate; the sinus was still slightly discharging. The patient refused all nourishment on that day on account of his inability to swallow; a throat culture was taken. Coincident with the appearance of these pharyngeal symptoms occurred graver constitutional disturbances; the pulse became rapid to 112, with a sudden rise in temperature which, on the first day of January, 1908, went up as far as 101, but dropped to 98 during the night; it then rose again the next morning to the time of the patient's death, which occurred at 3 p. m. on January 2. His physical symptoms during that time did not change.

The culture of the throat showed practically a pure growth of Loffler's bacilli.

At the autopsy, which was held on January 3, was found the following:

The soft palate and the uvula are pale oedematous and pushed up to the posterior nasal fossa; the peritonsillar structures are oedematous, but contain no membrane. There is no hypertrophy of the adenoid nasal tissue. On chiseling through the hard palate, the nasal mucus membrane appears free from any false membrane. The tongue is swollen, thick and pale, oedematous. The perilaryngeal tissues, especially in the epiglottidean fossa,

show greenish mucus membrane just above the mouth of the oesophagus. The tissues of the larynx are moderately oedematous without false membrane. Trachea and large bronchi are clear with the exception of being bathed in mucoid fluid.

Just below the lobe of the right ear, behind the angle of the jaw, there is a small sinus, from which exudes brownish fluid, which extends upwards and forwards into the anterior triangle of the neck and leads to broken-down necrotic cervical gland.

The left arm is much larger around the elbow than the right arm and boggy. The olecranon bursa and the elbow above it appear to be soft and fluctuating. On opening this, considerable pus escapes. The tissues of the forearm are oedematous and infiltrated with greenish pus. The subcutaneous fat and fascia necrotic; this extends to just above the elbow joint involving the olecranon bursa, but not involving the joint.

Apart from these lesions of greater interest to us here there were found a degenerative myocarditis, a purulent bronchitis, a hypertrophic interstitial splenitis, a degenerative and exudative nephritis and a degenerative parenchymatous hepatitis.

Cultures from the blood of the heart and from the spleen, and pus from the arm showed pure streptococci.

Sections were taken from the back of the tongue, the palatine tonsils, the lingual tonsils and the pharyngo-epiglottidean fold. In describing them I shall not attempt to present an exhaustive study of diphtheritic lesions in general, but shall recite only the more characteristic features of this case.

Section from back of tongue showed throughout coagulation necrosis of the epithelium in all stages, from the superficial to the deeper layers. Where it involved only the upper portions, the epithelium appeared primarily pale, glassy, swollen, with indistinct outlines, and vascular nuclei with progressive loss of chromatine. Such cells would coalesce, their protoplasmic material underwent coagulation necrosis and would lie passively on the top or on the side of less involved, or even normal, epithelium. Coincident with this necrosis there occurred marked dilatation of the superficial blood vessels extending into the epithelium, and an extravasation of blood and migration of leukocytes into the necrotic masses. All then became involved in the coagulation necrosis. Leukocytes were occasionally seen in places where necrosis of the epithelium had not become evident. Below the epithelium, which showed various stages of this process, the mucosa appeared markedly oedematous, the vessels much engorged, their endothelium proliferating, the walls in places hyaline, and surrounded by perivascular leukocytic inflammatory foci. The lymphoid channels were widely dilated, sometimes contained coagulated matter; the smaller lymph and tissue spaces filled with

leukocytes. The glands in the submucosa were for the greatest part quite intact, even in places where the coagulation necrosis of the epithelium had been complete, and inflammatory changes of the mucosa were pronounced. In places an entire loss of the lining epithelium was observed; the covering of the mucosa was here entirely made up of necrosed epithelium, leukocytes and blood which were loosely attached to the mucosa, with complete destruction of epithelium. Occasionally coagulation necrosis of the mucosa was observed. This ran along even under areas of well preserved epithelium. Where the necrosis of epithelium appeared old it showed disintegration of the coagulated masses into granular, or even fibrillar interwoven threads, forming a sort of network enclosing remaining coagulated masses of nuclei and blood detritus; occasionally these underwent a hyaline change. Fibrin could not be demonstrated in these necrotic patches, nor in the inflamed mucosa. In the epithelial cells, somewhat removed from the actual areas of beginning necrosis, mitosis could occasionally be seen. The superficial layers of all necrosing portions showed numerous bacilli of the Löffler type, but these were never found deep; where the change was quite recent, it could occasionally be seen that they reached down between epithelial cells which showed beginning necrosis. The most extensive loss of epithelium was found in the sections from the pharyngo-epiglottidean fold; there it had been removed entirely, leaving a more or less ragged, irregular, much inflamed mucosa without any covering at all; here also necrosis of the mucosa was found in places, occasional small clumps of streptococci were seen in these deeper layers.

The examination of the palatine tonsil showed oedema of the lymphoid elements, with irregular small areas of necrosis of the lymph cells, associated in places with the presence of streptococci. This was observed more especially in the neighborhood of the crypts, where it occasionally involved the epithelium; here also bacilli were absent and fibrinous exudation could not be demonstrated.

The lymphoid tissue of the back of the tongue was much changed. Here extensive areas of necrosis with loss of the lymphoid elements were seen, leaving only a hazy, fine reticulum which, however, gave no fibrin reaction, but enclosed a considerable number of polymorphonuclear leukocytes. In places, where the lymphoid tissue was somewhat better preserved, smaller areas of necrosis of these elements were seen; scattered through it were large clumps of streptococci only; there was no fibrinous exudation.

In shortly reviewing this case we have to deal with a severe erysipelatous infection with amelioration and exacerbation, later becoming more general.

As temperature and pulse chart indicate, the patient rallies from this when suddenly the new complication arises which, clinically, is well expressed in a sudden rise of temperature and pulse with new and severe throat symptoms.

Investigation shows two etiological factors: primarily the streptococcus, and secondly in the terminating lesion the bacillus of Löffler. Anatomically this terminating lesion consists of a recent coagulation necrosis of the surface epithelium, occasionally in the mucosa with an exudation free from fibrin. We cannot state whether this was owing to the early stage of the lesion or a particular feature of this infection. Interesting is the reversion of the usual course of events. More frequently, Löffler's bacillus is the primary exciting cause, and the streptococcus gains access after the way has been prepared for it. Here the attack for the syananche infection was undoubtedly prepared by the general streptococcus invasion, and the question arises whether this had primarily produced lesions in the throat allowing Löffler's bacillus to enter, or whether this entered *de novo*. It seems, from the fact that the bacillus was found in greater number in the more recent and superficial areas of necrosis, and that the streptococcus was not conspicuous, even in the actively inflamed mucosa, and that the course of events proceeded uniformly from above downward, that this particular terminating lesion is primarily the result of an invasion of Löffler's bacilli. In accord with that is the clinical picture which indicates that at a particular time a new interference in a favorably progressing disease had occurred.

It is instructive to know that the anatomical lesions found were really much more extensive than could have been anticipated from the clinical findings. If it is allowable to draw any conclusions from this, we may suspect that in cases of synanche in which the constitutional disturbances appear to be out of all proportion to the apparent local manifestation, a really greater involvement of these structures exists than appears evident from simple inspection.

Synanche complications are not infrequently observed in acute exanthemata, notably scarlet fever and measles. Occasionally they are found in other acute infectious diseases, as in typhoid fever. When they occur at the height or in the course of these diseases, it is difficult to determine what the importance of this infection is in relation to the original one. In the case under discussion, however, its occurrence and malignant influence appear well marked.

Judging from the records and the experience of others, the combination of erysipelas with synanche must be rare. In the extensive list of 220 cases collected by Councilman, Mallory and Pearce, a record of only one occurred in a child, and there the erysipelatous infection followed that of the synanche. Other text books do not mention even the possibility of this combination, and

for this, and the reasons previously discussed, I have thought it worth while to relate it.

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A CASE OF SYPHILITIC CIRRHOSIS OF THE SPLEEN, ASSOCIATED WITH GENERAL VISCERAL AMYLOID CHANGES, WITH A FEW GENERAL REMARKS ON THE NATURE OF AMYLOID.*

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This spleen, which is the seat of a rare but rather typical lesion, namely, a cirrhosis of syphilitic origin, came from a woman age 54 years, who died on the Medical Service of the City Hospital, after being under observation about two weeks. On her admission to the hospital she said she was a domestic, and her chief complaint was a vague stomach trouble.

Physical examination revealed a poorly nourished woman, who appeared markedly anaemic. Her teeth were very bad; tongue coated, with slight tremor. The heart was slightly enlarged, being beyond the nipple line; a systolic murmur was heard at the apex, and over the aortic area. The second aortic sound was accentuated. Pulse was full and regular. On palpating the abdomen the liver was felt a hand's breadth below the costal margin, its edge feeling firm, rounded. There was slight tenderness over the epigastrium. The abdominal veins were prominent. The rest of the examination was negative. The urine was amber, acid 1.010. Albumen, hyaline and granular casts were present. Blood count showed:

Erythrocytes	2,384,000
Haemoglobin	15%
Color index	0.3
Polymorphonuclears	87%
Large lymphocytes	4%
Small lymphocytes	9%

* From the Russell Sage Institute of Pathology. Presented to the New York Pathological Society, March 11, 1908.

While in the hospital her temperature was always about normal. Her pulse varied from 68 to 108, usually around 90. Her respirations were only increased at the time of death. Her blood pressure varied from 90 to 130. Her chief subjective symptoms were nausea, vomiting and diarrhoea. She gradually grew weaker, becoming irrational and incontinent. During the last few days there was a marked diminution in the amount of urine secreted. She died of air hunger (probably anaemic in origin).

The clinical diagnoses were:

Severe secondary anaemia—cause undetermined.
Cardiac hypertrophy.
Arterio-sclerosis.
Chronic nephritis.

Of her previous history, it is noteworthy that she had contracted syphilis when thirty years old, and received very little treatment. Three years later she had an operation of rectum (probably for stricture). When forty years old, she was in a hospital several weeks for a sypilitic iritis. One year ago patient was in City Hospital with a severe gastritis.

The summary of the autopsy findings were general amyloid infiltration of the spleen, liver, kidneys, adrenals, mesenteric glands and intestines. A syphilitic interstitial splenitis; chronic interstitial nephritis; chronic interstitial myocarditis; pulmonary oedema, and smooth atrophy of base of tongue.

The spleen is quite large; it weighed 870 gms. and measured 17x12x9 c.m. It was oval in shape and presented a notched anterior border. What impressed one at the autopsy was its great firmness. On section it cut with great difficulty, the knife encountering firm, cartilaginous masses, and areas of calcified tissue near the hilum; the cut section was a light hamred in color. The capsule is only moderately thickened and is free from a perisplenitis. What is most marked, however, is the rather unusual appearance of the splenic substance, especially near the hilum. Here the trabeculae are enormously thickened, being 2 to 5 to 10 m.m. across; they are a pale, bluish-white, and have a peculiar, hyaline semi-translucent appearance. These bands divide the spleen into small and large, irregular islands of the pulp. The large vessels, when cut transversely, show thick walls with a glistening appearance. The general color of the spleen pulp is light-red, but in places, near the trabeculae, a very dark red color is seen. The Malpighian follicles are hardly visible.

On staining with a dilute iodine solution, the surface presented numerous dark mahogany-brown points, most of which represented the regions of the arteries of the Malpighian corpuscles.

Section stained with methyl violet showed a diffuse amyloid infiltration, a rose-red, homogeneous substance appearing in and around the arteries of the Malpighian follicles, in the capillaries of the spleen pulp, and in the walls of the cavernous sinuses. The deposition of the amyloid material is rather irregular; although diffuse, it is not massive.

Sections stained with Eosin-haemotoxylin and Mallory's anilin blue methods show enormously thickened trabeculae, which are composed of a poorly nourished, not cellular, hyaline connective tissue. The reticulum supporting the spleen pulp is greatly thickened, and there is a great waste in the splenic substance. There is also marked atrophy of the lymphoid mantels, the central artery appearing bare in many places.

In other words, we have to deal here with an extensive cirrhosis of the spleen. If we look for the cause there can probably be no question that it is of syphilitic origin, for in favor of this is the extent and distribution of the lesion; the presence of thick, isolated bands of connective tissue similar to those seen in syhpilitic cirrhosis of the liver, these bands being most marked around the hilum; the appearance of the tissue itself; the affections of the blood-vessels; the amyloid degeneration, which, in this case, must be attributed to syphilis, and the certainty of syphilis as an etiological factor.

The liver is quite large, its size being due mostly to an increase in its vertical diameter. It is very pale in color, appearing decidedly anaemic; quite firm and solid, it has a peculiar india-rubber consistency, namely, a pitting on pressure which disappears when the pressure is removed. The anterior free edge is firm and rounded. Cut section is very pale and has a glistening, translucent appearance. There can be made out a coarse pink network, which in places can be said to represent the middle and outer zones of the liver lobules, but in most cases the anatomical relation of the liver lobules is indistinct. The cut surface stained with a dilute iodine solution shows a rich mahogany-brown network, which replaces the pink network above described. Microscopically, almost all the capillaries are replaced by a homogeneous mass; the liver-cells are not at all prominent, and the radiating appearance of the liver lobules is replaced as it were by these swollen, homogeneous capillaries. The lumen of the capillaries cannot, as a rule, be made out, and embedded in these homogeneous masses are seen scattered capillary endothelial cells. The liver cells show all stages of atrophy and degeneration. This is due both to the pressure of the amyloid substance and to the diminution of blood supply. The connective tissue in the periportal spaces is well preserved. What is remarkable is the fact that there is no replacement of connective tissue due to the destruction of the liver parenchyma; the fibrous tissue is undoubtedly too poorly nourished to proliferate. The process here represents a severe

amyloid infiltration. This has reached such a late stage that it is impossible to make out primary changes in the small branches of the hepatic artery.

The left kidney is very small. It presents a rough, granular surface and is quite pale. The capsule strips with difficulty, leaving a rough, nodular surface, and in places tearing out portions of the kidney parenchyma. On section the kidney cuts very firmly. The cortex is irregular, thick and thin, but on an average thinner than normal. It is a pale putty color; the Malpighian bodies appear as pale, glistening, semi-translucent points, but the glomerular rows are in many places broken. The straight vessels in the cortex also show as glistening, hyaline-like lines. The pyramids, in contrast to the cortex, are red, straight vessels, in places prominent. On staining cut surface with a dilute solution of iodine, most of the glomeruli, and many of the straight vessels, stand out as prominent, dark mahogany-brown streaks.

The right kidney presents similar appearances. Microscopical examination shows marked chronic interstitial nephritis, with atrophy of parenchyma, and marked amyloid infiltration of the capillaries, of the glomeruli and of the straight vessels.

The adrenals are large and present a few cortical adenomata. The medulla contains considerable amyloid, almost entirely replacing the medullary cells. The intestines show a marked amyloid infiltration of the vessel walls.

I may be allowed to add a few remarks about the nature of amyloid. It has been held that amyloid may be formed in three ways: first, by degeneration of parenchyma cells, or interstitial tissues; second, by a precipitation from the tissue juices, and third, by a combination of the first and second; part of the substance being furnished by degeneration and part of it by precipitation from fluids.

Virchow[1] thought that the parenchyma cells played an important role in the process. He describes them as being infiltrated and later replaced by amyloid material.

He regarded amyloid as a substance derived from proteid, but not necessarily of the same nature, which is deposited in the tissues, under certain conditions, as a homogeneous, waxy mass, but occurs in the serous cavities as round or oval bodies which are laid down in concentric layers. He held that the substances producing amyloid were in the circulating blood, chiefly because the amyloid began in so many different viscera at the same time, and progressed diffusely.

In speaking of amyloid degeneration of the liver, he says: "If a single hepatic cell be watched, its previous granular contents, which gives

every hepatic cell its cloudy appearance, is seen gradually to become homogeneous, the nucleus and cell-wall gradually disappears, and at last a stage sets in in which nothing more can be perceived than a homogeneous shining body * * * in this manner * * * the whole of the hepatic cells become converted into amyloid flakes."

Virchow, therefore, thought that amyloid infiltrated the parenchyma cell, an observation which I cannot confirm in any of the sections from this or similar cases. The parenchyma cells appear to me to be affected only in so far as they atrophy or disintegrate from the pressure or malnutrition, caused by the presence of the intercellular amyloid substance.

But in the amylaceous bodies, such as are found in the prostate, brain, and lungs, the degeneration of the parenchyma cells is evident; for instance, in the corpora amylacia of the prostate it has been found that the cells lining the prostatic acini undergo a colloid degeneration, and such cells are even seen in the centres of these bodies. The periphery is formed by the precipitation of crystaline amyloid which is derived from the tissue juices.

Siegert still holds that amyloid, in general, bears a close relation to the degenerating parenchyma cells, while Ziegler[2] and Stillung[3] hold this view only so far as the amylaceous bodies are concerned.

From a careful study of the sections in this case, I can only hold Wichman's[4] view, namely, that there is no evidence that amyloid (excluding amylaceous bodies) is formed by degeneration of either parenchyma cells, basement membranes, or intercellular substance, but that it is derived rather from an infiltrating albumen.

As to the nature of the formation of amyloid, Czerny[5] believes that it takes origin within the leucocytes. He produced artificial suppuration, and observed that the leucocytes, when stained with iodine, showed brown granules (iodophilia), which turned blue when treated with sulphuric acid. He believes, that this pre-amyloid material passes into the tissues and later becomes amyloid. It is impossible for me to conceive that the enormous amount of amyloid occasionally present in the viscera in this disease is derived from such a source, since in iodophilia the absolute number of granules is small.

Krawkow[6] states that amyloid changes cannot occur from aseptic suppuration (such as caused by turpentine), but that the presence of microbes is absolutely necessary, and that the absorption of their poisons play an important part.

Chemical advances have added greatly to our knowledge. It is well known that amyloid is an amorphous substance formed by the combination of chrondroitin sulphuric acid and an alkaline proteid (probably of the nature of a liston).

But there exist marked chemical differences between various amyloids, which must be attributed to varieties in the histon radicle.

I believe that the nature of amyloid disease, which demands this combination of a histon-like proteid and chrondroitin sulphuric acid* may be explained in this way: Under certain conditions there is present in the blood a large quantity of a definite proteid which is derived either from a great destruction of leucocytes, as in chronic suppurations, especially in cavities; or from waste of lymphoid cells as in syphilis. This proteid, which is of the nature of an alkaline histon, combines with the chrondroitin sulphuric acid, which, as evidence indicates, is already normally present in the capillary walls, and in other organs, and progressively precipitates in the form of amyloid.

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*Chrondroitin sulphuric acid is an etherial sulphuric acid of the formula $C_{18}H_{27}NSO_{17}$. It was prepared pure by Mörner from cartilage; it has also been found in the intima of the aorta, cortex of kidney, bladder, ligamentum nuchae, bone substance, and in amyloid livers. It easily breaks down into chrondroitin and sulphuric acid $C_{18}H_{27}NSO_{17}+H_2O=H_2SO_4+C_{18}H_{27}NO_{14}$. Chrondroitin is a monobasic acid with properties similar to gum arabic. Chrondroitin further breaks down into chrondroisin $C_{12}H_{21}NO_{11}$. Chrondroisin is a gummy substance.

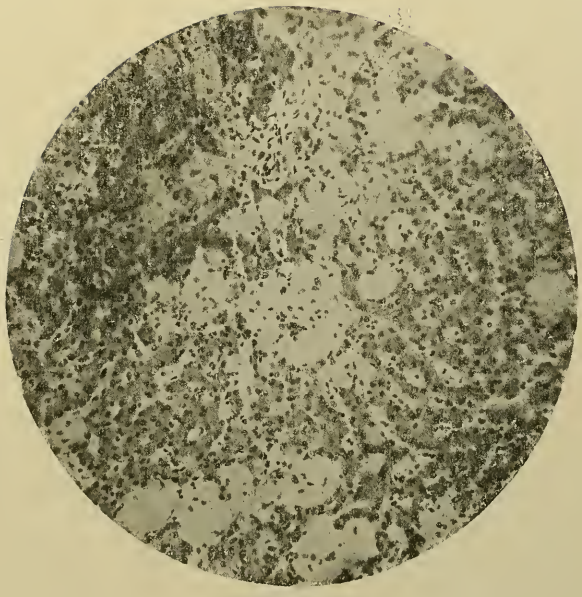

Fig. 1. Amyloid infiltration in the liver with simple atrophy of liver cells.

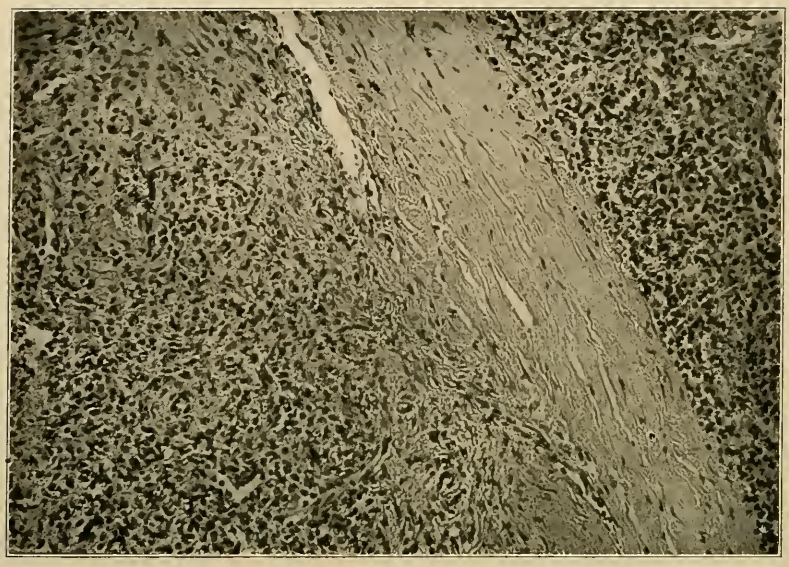

Fig. 2. Syphilitic scar in the spleen.

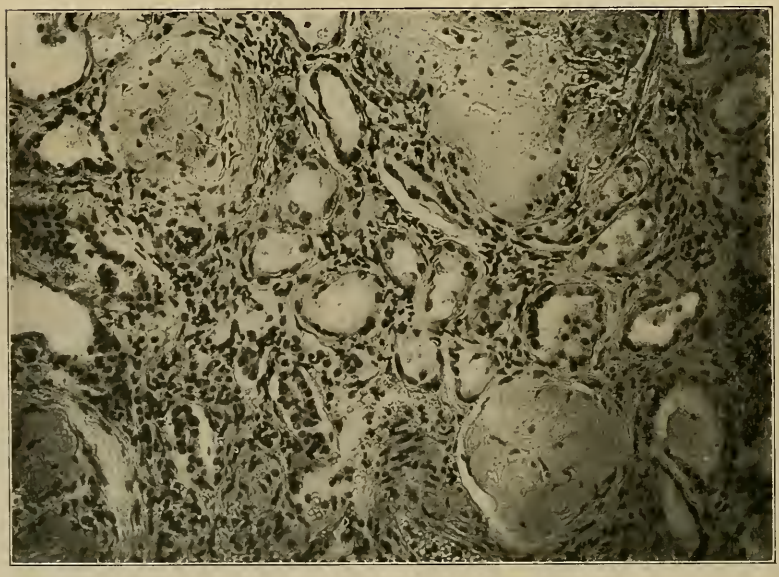

Fig. 3. Amyloid infiltration of glomeruli in kidney with simple cellular atrophy.

THE PATHOLOGICAL FINDINGS IN EIGHT CASES OF GENERAL SEPSIS FOLLOWING CHRONIC SUPPURATIVE MIDDLE EAR DISEASE, OBSERVED DURING THE YEAR 1907.*

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During the last year there have been autopsied in this institute, eight cases of sepsis due to middle ear disease, some of which were clinically quite obscure and sufficiently complicated to make their origin difficult to trace. They are presented here largely for two reasons—first, chronic middle ear disease, although of long standing, and latent, may be the cause of a general sepsis; secondly, it shows how careful even an anatomical examination must be made before the diagnosis of cryptogenetic septicaemia is justifiable.

Clinically, some of the cases presented obstacles which were difficult to overcome, largely on account of the lack of objective and subjective symptoms pointing towards the original focus.

Case I.—Female; Age, 28 Years.

Summary of Clinical History:

Patient was admitted through Bellevue Hospital, where she received no medical attention, but was transferred to the City Hospital with the diagnosis of chronic alcoholism and a possible epilepsy—no history accompanied the patient.

On admission to the reception ward she was unable to give any account of herself. Her temperature was 100.3 F., pulse 128 to the minute, and respirations 32. Almost immediately after entrance she had a convulsion, which lasted two minutes, and during which her head and mouth were drawn to the left. She remained unconscious for ten minutes. Two hours later she had a similar seizure lasting five minutes; her pulse had now become very weak, and in spite of stimulation she died five hours after admission to the hospital.

Clinical diagnosis: Uraemia?

Anatomical diagnosis: Brain abscess; chronic purulent mastoiditis, and otitis media; acute parenchymatous degeneration of kidneys, heart and liver; pulmonary oedema.

Brain: On removing the calvarium the vessels of the dura are distended with blood. The surface of the brain is dry, and there is no ap-

* From the Russell Sage Institute of Pathology, New York City Hospital.

parent increase in intra-cranial pressure. On removing the brain, about 5 c.c. of pus escapes from the right tempero-sphenoidal lobe; this is seen to escape from a small opening in the third auditory convolution. The cortex around this opening is greenish in color. On opening the tempero-sphenoidal lobe, a small oval cavity is seen, of about 8 c.c. capacity; this cavity is well walled off from the surrounding brain tissue, and occupies about the middle of the tempero-sphenoidal lobe. It is separated by about 1.5 c.m. of normal brain substance from the roof of the tympanum.

Temporal Bone: The mastoid cells are almost entirely obliterated by bone, which has an ivory consistence. The antrum is filled with pus and granulation tissue. The roof of the tympanum is thin and necrotic. No sinus thrombosis.

Cultures made from the ear showed streptococci and staphylococci.

Case II.—Male; Aged 18 Years.

Summary of Clinical History:

This boy entered the Manhattan Eye and Ear Infirmary on account of an acute mastoiditis; he soon became comatose. A mastoid operation was performed, and during the operation a trephine opening was made into the middle fossa of the cranium, as a brain abscess was suspected. The patient before the operation had had double choked discs. The entire tempero-sphenoidal lobe was explored with a Grafe knife, without making a dural flap, but no abscess was found. His coma disappeared after the operation, and his cerebral condition was then supposed to be due to a serous lepto-meningitis. While convalescing, facial erysipelas developed (two months after the operation). He was later sent to the City Hospital with a recurrent facial erysipelas (three months after the operation).

His second attack of erysipelas was not severe, and he was practically well, when his temperature suddenly rose to 102.4 F., pulse 92, and respirations 24. The mastoid wound had healed, and he presented nothing on examination but a double optic neuritis. Lumbar puncture showed turbid fluid under pressure and containing 93 per cent. poly-morphonuclears, no organisms. He died soon after this last examination.

Clinical diagnosis: Acute meningitis, due to middle ear disease.

Anatomical diagnosis: Brain abscess; acute parenchymatous degeneration in heart, kidneys and liver.

Brain: Over the right mastoid region is a curved healed incision scar; a small portion of its middle is still open and exudes on pressure a slight amount of pus. On removing the calvarium the dura is quite adherent to the left tempero-sphenoidal lobe, the brain is dry, the vessels of the pia distended with blood. The left tempero-sphenoidal lobe is greenish, soft and on

section a large cavity is found, holding about 30 c.c. of pus. This cavity is well walled off from the surrounding brain substance and occupies the greater part of the centre of the lobe. It is separated from the trephine opening by considerable cortex.

Pus from the abscess showed streptococci.

Case III.—Male; Aged 48 Years.

Summary of Clinical History:

Patient came into the hospital complaining of pain in back, cough and chilly sensations. For the last six weeks he had been drinking whiskey steadily, beginning in early morning and remaining drunk for the remainder of the day. For the last two weeks he has had a racking cough, morning vomiting and loss of appetite. Other points in history are unreliable and worthless.

The patient is a poorly nourished man with typical alcoholic features, dilated cutaneous capillaries, etc. Lower eyelids are puffy and oedematous. Lungs are emphysematous and a few dry rales are heard over the bases posteriorly. Liver below the costal margin. Urine contains large quantity of albumen, hyaline and granular casts, and a large amount of pus. For the first three days after admittance he ran a temperature of 98-102 F., and had a little cough; his temperature soon came down to normal, where it staid at 98-99 F., until death.

He was thought to be recovering from a long abuse of alcohol, and in addition to have a nephritis with a probable cystitis.

Eight days after admission he complained of a discharge from his left ear; examination showed an old perforation of the left drum membrane, with a rather profuse discharge. No mastoid tenderness. Smear from pus showed pneumococci. Leucocyte count was 24,000. Patient was running practically no temperature, and ear was syringed out and treated. The discharge from the ear soon ceased. Six days after the discharge was noticed, patient suddenly became cyanotic and got up a pulmonary oedema; in four hours he was comatose, with typical uraemic convulsions—temperature sub-normal, he soon died.

Diagnosis: Uraemia.

Anatomical diagnosis: Pyaemia; chronic suppurative mastoiditis; metastatic (haematogenous) cortical kidney abscesses. Acute parenchymatous degeneration in all viscera.

Left Kidney: Weight, 300 gm. The kidney is large, globular, and is soft and apparently fluctuates. It measures 13 x 7 x 5 cm., and is grossly irregular. On section the capsule strips with slight difficulty and is thin and vascular. The surface of the kidney beneath is pale, reddish-brown,

with numerous dirty-white, elevated, small and large masses; in many places these are confluent, forming whitish elevations some 3-4 mm. in diameter. The stellate veins are prominent, giving the surface a dark-reddish color, but on close observation the kidney substance is pale. On cut section the markings are irregularly destroyed, but the cortex can be made out as a dark-red swollen zone, some 1 cm. in thickness. The cortex contains numerous white, granular areas, which have a soft pulpy surface. The pyramids are injected. The pelvis and the ureter is free. The right kidney presented a similar appearance.

Brain: All the vessels are distended with blood, the white matter has a diffuse pinkish color.

Temporal Bone: The lateral sinus is not thrombosed. The mastoid cells are filled with greenish pus, the cells necrotic.

Culture from the ear show pneumacocci.

Smears from the kidney showed a capsulated diplococcus.

Case IV.—Male; Aged 48 Years.

Summary of Clinical History:

This man entered the hospital in the evening because he had been drinking for the last three weeks, and he thought he was on the verge of delirium tremens. He had on admission a slight discharge from the ear, no pain or tenderness over the mastoid, but rheumatic pains around the ankles. On account of the change in staffs in the hospital at this time, he was transferred to a new house physician the next morning, as an alcoholic with vague rheumatic pains, and he was not carefully looked over until the next morning, some 40 hours after admission, when the following notes were made: Patient has been irrational at times, this is becoming constant, he has a coarse tongue tremor and constantly picks at the bed-clothes, his physical examination is negative except for the following: Temperature 100.1 F., pulse 78, and respirations 22 to the minute. No mastoid tenderness, but a slight discharge from one of his ears was noted. The right ankle was swollen, red and oedematous. Urine contained no albumen, but hyaline and granular casts. Leucocytes 22,000; polymorphonuclears 90 per cent. Pus from ear showed staphylococci.

Immediate operation was advised and revealed a chronic suppurative mastoiditis, a large thrombosis of the lateral sinus and pus in the right elbow and ankle joints.

Anatomical diagnosis: Pyaemia (streptococcus found in heart), sinus thrombosis of lateral sinus; multiple pus joints: acute parenchymatous degeneration in all viscera.

Case V.—Male; Aged 43 Years.

Summary of Clinical History:

This patient was convalescing from a severe delirium tremens when he complained of pain and deafness in one of his ears. An acute exacerbation of a chronic suppurative otitis media was diagnosed, and a paracentesis was done; he remained two months in the ward, complaining of nothing but a continuous discharge from his ear. His temperature would rise every four or five days to 100-101 F., in the intervals it would remain about normal. He took to his bed and was one day found with a temperature of 102.2 F., examination revealed at this time consolidation in both lungs and tubercle bacilli were found in his sputum. He also had cough and night sweats. He became delirious on the next day, his leucocyte count was now taken and found to be 17,000. Exacerbation of otitis media was suspected, and a diagnosis of brain abscess was made, he was trephined over the middle fossa and explored for abscess, but none was found. He died soon after the operation.

Clinical diagnosis: Acute miliary tuberculosis or sepsis from the ear condition.

Anatomical diagnosis: Acute purulent meningitis; acute encephalitis; purulent mastoiditis and otitis media with erosion of the roof of the tympanum; parenchymatous degeneration in all viscera. Pulmonary tuberculosis.

Case VI.—Male; Aged 60 Years.

Summary of Clinical History:

Two weeks before admission patient was suddenly taken with a Bell's palsy; he also had pain, deafness and a slight discharge from the ear of the same side (right). Examination showed a chronic otitis media, with an old perforation in the drum membrane, and polypi in the middle ear. An operation was done and the mastoid cells were found infiltrated with pus; granulation tissue and polypi were removed from the middle ear. The day after the operation the patient had a chill, followed by a rise in temperature to 103 F. The ear wound was clean. There was an area of dullness with broncho vesicular breathing over the base of the left upper lobe. His temperature gradually dropped to normal. Eight days after the operation, he again had a rise in temperature to 103 F., no cough, no pain, but he had the same area of dullness over the left lung, and besides a few rales in the right axilla. His respirations were shallow, and his general condition poor. He developed pleuritic rales over left base and dullness over both apices. Leucocytes 13,200; polymorphonuclears 87 per cent. He died soon after these last notes were made.

Clinical diagnosis: Broncho-pneumonia.

Anatomical diagnosis: Extensive septic broncho-pneumonia with cavity and abscess formation; chronic purulent mastoiditis; parenchymatous degeneration in all viscera.

Lungs: Left lung large, its anterior border grayish, creptant except for a small portion of the upper lobe near its anterior border, which is firm and red. Pleura at the apex contains a few puckered pleural adhesions. The interlobar fissure is obliterated in places by fresh adhesions, and on separating shows an area on the upper lobe which is greenish and contains irregular yellowish areas; this area fluctuates. Over the lower lobe the pleura shows fresh exudate. The lung beneath this is dark red. On section the lower lobe is firm, but contains considerable air; it is grayish in color. Near the posterior border, and about the middle of the lobe are a few large, raised cavities which contain purulent matter. The upper lobe on section presents, near the interlobar fissure, a dark reddened area, about 2 x 3 cm. which is firm and devoid of air. Around this area the lung substance contains irregular, riddled cavities, all of which are filled with pus. The right lung is similar to the left in appearance.

Brain: The brain shows congested vessels. Over the roof of the tympanum is a small, fluctuating, purulent mass, which lies epi-dural in position. The bone beneath this mass is necrotic. The lateral sinus is clear.

Case VII.—Female; Aged 27 Years.

Summary of Clinical History:

While convalescing from a left inguinal colostomy, which was done for a syphilitic ulceration of the rectum, the patient complained of pain in left ear. On examination the drum membrane shows an old perforation, there is a purulent discharge from the ear, but very slight mastoid tenderness. Her temperature was 102 F., an operation was advised, and performed, a complete curettage of the purulent mastoid cells was done. She had had the temperature several days before the operation was performed, her fever being thought to be due to her colostomy wound. She soon developed pain in abdomen, and a septic right shoulder joint. The patient in the start was in very poor physical condition, and she soon became too weak to operate on a second time; she died with a typical picture of general sepsis.

Anatomical diagnosis: Pyaemia; acute parenchymatous degeneration in all viscera; acute suppurative peritonitis; purulent arthritis of right shoulder. Necrotic tympanum roof. Cultures showed streptococci. The peritonitis had no connection with the colostomy wound.

Case VIII.—Male; Aged 25 Years.

Summary of Clinical History:

Patient enters hospital with a chronic otitis media. Examination showed an old perforation in the drum membrane of the left ear, with a profuse discharge, and mastoid tenderness. Temperature 100 F., pulse 100, and respirations 24 to the minute. Leucocytes 11,000. The next evening his temperature had risen to 102 F., pulse 90 and respirations 24. The morning of the following day the temperature was down to 100.8 F., but in the evening again it was over 103 F. He ran this septic type of temperature for five days, when the following notes were made:

Patient appears stupid and rolls his head as if he had great pain. The discharge from the ear has subsided, and he has only slight tenderness over the tip of the mastoid. Leucocytes 21,000; polymorphonuclears 89.5 per cent.

Operation on the mastoid was performed. The mastoid tip was found necrotic with extensive involvement of the cells. The lateral sinus was not involved. After the operation, his temperature still remained at 103 F. Forty-eight hours after he became comatose and he soon died of pulmonary oedema.

Lumbar puncture just before death showed a purulent fluid under pressure and containing streptococci.

Anatomical diagnosis: Acute lept-meningitis; encephalitis; spinal meningitis; acute parenchymatous degeneration in all viscera.

Brain: On removing the calvarium, there is marked increase in the intra-cranial pressure, the convolutions being flattened and the brain surface appearing smooth. Over the left Sylvian fissure, and extending along the course of the brain in this region, appears a yellowish and turbid exudate, which is sub-pial. The entire base of the brain is covered with a greenish-yellow exudate which mats together all the structures at the base. The spinal cord shows a purulent meningitis over almost its entire length.

Temporal Bone: The cranium contains two small epidural abscesses, one lying over the apex of the petrous portion of the temporal and just below the roof of the tympanum; the other over the exit of the eighth nerve.